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THE

# MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—9TH YEAR. SYDNEY: SATURDAY, SEPEMBER 30, 1922. No. 14.

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## BREAST FEEDING.[1]


BY MARGARET HARPER, M.B., CH.M. (SYDNEY),

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DR. MACDONALD GILL AND DR. PARKER are giving us the benefit of their experiences in the treatment of the nutritional disorders of infancy and I thought it would be useful if I introduced the subject of the prevention of these conditions.

Two great causes of malnutrition are (*i.*) improper feeding, (*ii.*) infections, such as summer diarrhœa.

Correct methods of feeding are then most important in the prevention of these conditions and most important of all methods is breast feeding.

It is generally admitted that one of the principal means of reducing the infant mortality rate, as well as the morbidity rate, consists in promoting sound ideas on breast feeding. To quote Marfan: "For this propaganda the essential agent and the most indispensable is the doctor. In spreading the necessary knowledge, in correcting errors and combating prejudice no one can take his place. Nothing is of such value as the advice he gives in each particular case, whether in his private practice or in infants' consultations." And here Marfan adds a sentence which we in Sydney might lay to heart. He says: "In order to fit him to fulfil this task it is necessary that he should be taught during his course of study the hygiene and the pathology of the infant and particularly the science and art of infant feeding." It will be generally admitted that it is owing to the lack of such training of our students that many mothers fail to suckle their infants.

Under modern conditions of strain, domestic and other, difficulties do arise which may interfere with the satisfactory feeding of infants by their mothers, especially if there is no one at hand who understands the details and recognizes the importance of breast feeding. If we study the difficulties carefully, we almost always find that they are surmountable. The failure of the mother to feed her baby at the breast is not as a rule due to lack of will on her part, but to lack of knowledge, to over-anxiety and to readiness to believe that the milk does not agree with the infant.

The great majority of the difficulties are due to bad management in the first few weeks of life. The baby becomes cross and the mother discouraged and soon the breast feeding is a thing of the past.

The most important points in establishing and

[1] Read at a meeting of the Section of Pædiatrics of the New South Wales Branch of the British Medical Association on August 18, 1922.

maintaining a satisfactory supply of breast milk are:

(1) The regular stimulation of the breasts by the sucking of the infant at proper intervals.

In most cases the three-hourly interval from birth, with the long interval of eight hours at night, is most successful. In some cases where the supply of milk is plentiful and the infant strong and lusty the four-hourly interval answers best. The first week of the infant's life is a time of adaptation, both for the mother and child, but usually, if properly handled, at the end of that time the infant has fallen into the habit of wakening at the proper intervals for its food. As an objection to the formation of the habit of regular feeding, it is often said that mothers of savage races and females of animals suckle their young, following no rules, merely according to instinct. But at the present time women live under such conditions that, unless a proper amount of time for rest and for other duties can be obtained, they will certainly not be able to fulfil their natural duty of suckling their babies. The baby is more placid and contented if fed regularly and not too frequently. We must recognize the evils which result from civilization and combat them as we can.

(2) The use of the complementary feeding instead of the supplementary in cases where the supply of breast milk is either temporarily or permanently insufficient. By complementary feeding is meant the addition of a certain amount of artificial food to each breast feeding that is insufficient. Supplementary feeding is the replacing of one or more breast feedings with artificial food. The latter method should never be used if it is desired to keep up or to increase the supply of breast milk. The lack of regular stimulation is sure to lessen the supply of milk. This is indeed the best means of starting weaning.

When it is realized that every drop of mother's milk is of value to the infant, the advantage of maintaining even a small supply is evident.

The milk of every animal is specific for its own young and, although the special vital properties are not well understood, clinical experience shows clearly that even a small amount of breast milk will so act on the infant's metabolism, either by aiding in the digestion of the artificial food or in some other way, that a gain in weight will occur. Once started, provided the artificial food is of a suitable kind, it will continue, even after the breast milk is no longer given (see Charts III. and IV.). The question then arises as to how we are to know the amount of complementary feeding required.

If the baby is not gaining in weight, is hungry and proper weighing scales are not available, we have to depend on the infant's instinctive appetite. We may tell the mother to offer the baby so much of a milk mixture at the end of each breast feed and trust that the baby will only take what it needs. Watch the condition of the infant and arrive at conclusions. This method is rather haphazard and, although it may be successful, wherever it is at all possible, the amount of breast milk obtained should be determined by means of the test feed.

The baby is weighed before and after it has been put to the breast and the amount of milk it obtains is found. If this is insufficient, a complementary feeding of a calculated amount is given. The use of this method of ascertaining the quantity of food taken by the infant has enlightened us in many ways. It has shown that the normal, thriving baby gets meals of varying size during the day.

It has also shown that the quantities taken by the infant do not correspond with the anatomical size of the stomach. Cantley gives the capacity of the infant's stomach as follows:

At birth, 30 cubic centimetres (one ounce); at the end of the first month, 60 cubic centimetres (two ounces); at the end of the second month, 75 cubic centimetres (2.5 ounces); at the end of the fourth month, 90 to 120 cubic centimetres (3 to 4 ounces); at the sixth to ninth month, 150 to 180 cubic centimetres (5 to 6 ounces).

The following quantities were taken by babies at "Tresillian":

AVERAGE QUANTITIES FOR ONE WEEK.

*Age: Two to Three Months.*

| | | |
|---|---|---|
| 6 a.m. .. .. | 216 cubic centimetres = | 7.6 ounces |
| 10 a.m. .. .. | 156 cubic centimetres = | 5.5 ounces |
| 2 p.m. .. .. | 128 cubic centimetres = | 4.5 ounces |
| 6 p.m. .. .. | 125 cubic centimetres = | 4.4 ounces |
| 10 p.m. .. .. | 139 cubic centimetres = | 4.9 ounces |
| Daily Average | 784 cubic centimetres = | 27.6 ounces |
| Largest Quantity at any one feeding | 256 cubic centimetres = | 9 ounces |

*Age: Three to Four Months.*

| | | |
|---|---|---|
| 6 a.m. .. .. | 170 cubic centimetres = | 7 ounces |
| 10 a.m. .. .. | 128 cubic centimetres = | 4.5 ounces |
| 2 p.m. .. .. | 162 cubic centimetres = | 5.7 ounces |
| 6 p.m. .. .. | 150 cubic centimetres = | 5.3 ounces |
| 10 p.m. .. .. | 162 cubic centimetres = | 5.7 ounces |
| Daily Average | 810 cubic centimetres = | 28.5 ounces |
| Largest Quantity at any one feeding | 261 cubic centimetres = | 9.2 ounces |

*Age: Four to Five Months.*

| | | |
|---|---|---|
| 6 a.m. .. .. | 230 cubic centimetres = | 8.1 ounces |
| 10 a.m. .. .. | 193 cubic centimetres = | 6.8 ounces |
| 2 p.m. .. .. | 168 cubic centimetres = | 5.9 ounces |
| 6 p.m. .. .. | 179 cubic centimetres = | 6.3 ounces |
| 10 p.m. .. .. | 168 cubic centimetres = | 5.9 ounces |
| Daily Average | 971 cubic centimetres = | 34.2 ounces |
| Largest Quantity at any one feeding | 340 cubic centimetres = | 12 ounces |

The first feed after the long rest is the largest in the day. The infant seldom needs a complementary feeding at this time.

We also learn from the test weighing that an infant who is on three-hourly feeding, when the interval is lengthened to every four hours, gets in the twenty-four hours practically the same quantity of milk, taking larger meals.

The improvement which so often occurs in the comfort and general well-being of an infant when it is fed less frequently, is evidently not due to lessened quantity of food, but to the longer rest given to the stomach and to the complete digestion of one meal before the rest is added.

In order to estimate the amount of complementary food required, the most accurate method is to weigh before and after each feeding and to complement according to the amount obtained. But this is often impossible and quite satisfactory results can be obtained by giving a test feed at different hours on two or three consecutive days. From this an estimate

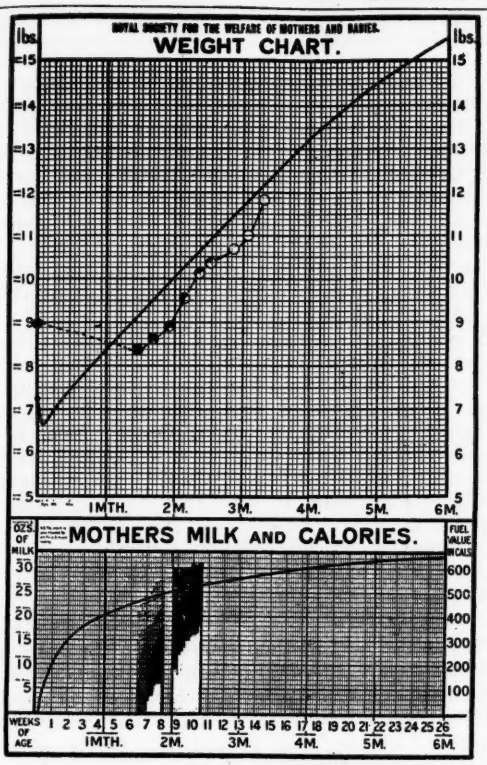


CHART I.—History of case in notes. Establishment of full breast feeding after six weeks of artificial feeding. Black represents caloric value of artificial food taken. White represents mother's or foster mother's milk.

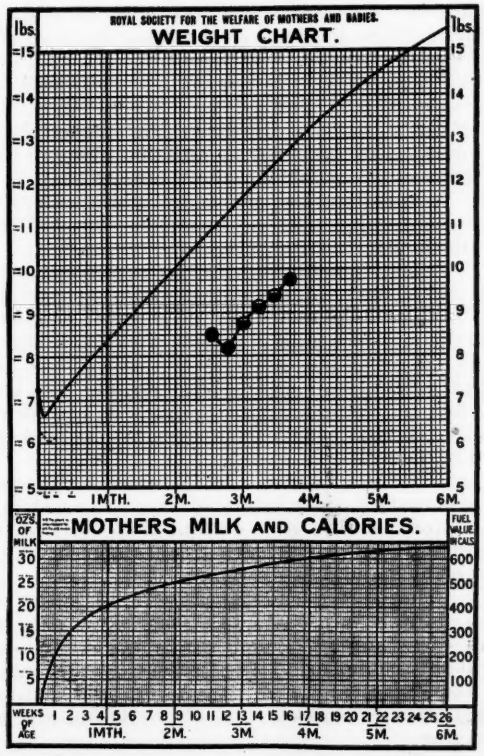


CHART III.—Showing value of small quantity of breast milk given in addition to artificial food. This infant was under observation and careful dieting for five weeks without any gain in weight. Then 150 cubic centimetres (five ounces) of breast milk replaced a like quantity of artificial food daily with immediate

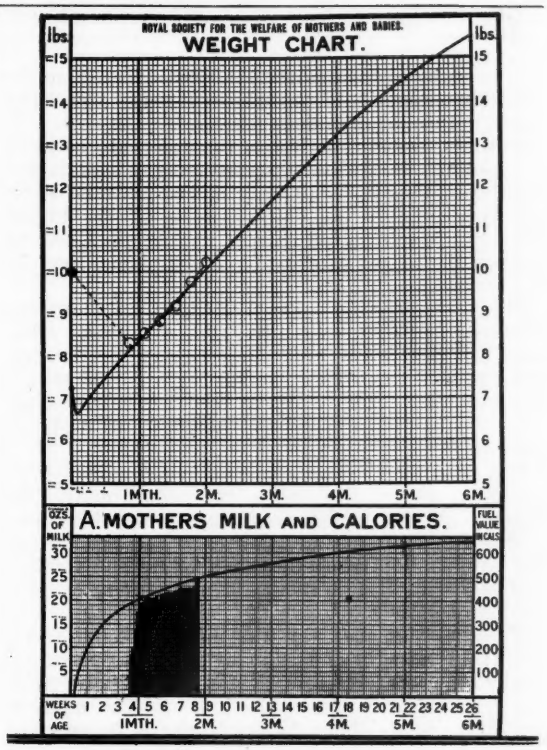


CHART II.—Showing how the milk can be increased by artificial stimulation only. The infant owing to malformation of mouth unable to suck. Methods used: Hot and cold sponging, massage, digital expression. The lowest chart (B) shows total quantity of milk produced by mother. Quantity not required by her own infant was used for others. Upper chart (A) shows her infant's weight and the quantity taken by him.

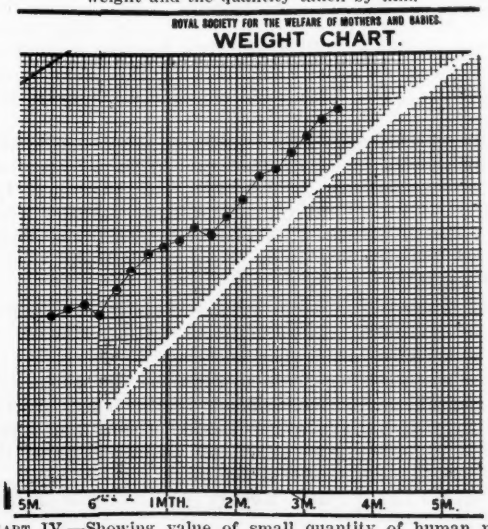


CHART IV.—Showing value of small quantity of human milk. Although this infant was getting a large number of calories in artificial food, he did not gain in weight until thirty cubic centimetres (one ounce) of artificial food in each feeding was replaced by human milk. Black represents artificial food.

may be made of the amount required. Here also the instinctive appetite of the baby will help. Generally it will refuse what it does not need.

Mothers are apt to lessen the intervals between the feedings if the baby seems hungry and is not gaining in weight. This only makes matters worse and very quickly the baby will be off the breast entirely. Much better results will be obtained by keeping the longer intervals and complementing the breast feedings.

The accompanying chart (see Chart I.) shows the establishment of breast milk after six weeks. The baby was put to the breast once a day for the first three days of life. Then the mother was told that it was useless for her to try to feed her baby, as the food she took would go to "nourish her and not the milk."

The food then given was condensed milk. This did not agree and "Lactogen" was substituted. The "Lactogen" was mixed according to the directions on the tin, with the addition of sugar and butter. The baby had diarrhœa on this mixture and vomited its food. This sort of history is, unfortunately, quite common.

On admission the baby was thin and flabby, with loose folds of skin and extensive excoriation of the thighs and buttocks. A few drops only of milk could be expressed from the breast. The methods used in re-establishing the milk were as follows: A few drops were expressed at the first massage and hot and cold sponging were started and the baby put to the breast regularly. On the fourth day the baby sucked 17.5 cubic centimetres and 23.5 cubic centimetres were expressed. Complementary feedings up to 120 cubic centimetres were given.

As the milk gradually increased the baby sucked at 6 a.m., 85 cubic centimetres (3 ounces); at 9 a.m., 39 cubic centimetres (1⅜ ounces); at 12 noon, 14 cubic centimetres (½ ounce); at 3 p.m., 7 cubic centimetres (¼ ounce); at 6 p.m., 7 cubic centimetres (¼ ounce); at 9 p.m., 7 cubic centimetres (¼ ounce). Later: At 6 a.m., 170 cubic centimetres (6 ounces); at 9 a.m., 64 cubic centimetres (2¼ ounces); at 12 noon, 39 cubic centimetres (1⅜ ounces); at 3 p.m., 39 cubic centimetres (1⅜ ounces); at 6 p.m., 64 cubic centimetres (2¼ ounces); at 9 p.m., 28 cubic centimetres (1 ounce). Here the first feeding needed no complementary food. The rest of the meals were complemented to 150 cubic centimetres (about five ounces).

The mixture was cows' milk, sugar of milk and boiled water, with no added fat. This was equivalent to carbo-hydrate, 6.9; fat, 1.4; proteins, 1.4. Each litre would therefore yield 468 calories (*i.e.*, 13.3 calories to the fluid ounce). The breast milk yielded 700 calories to the litre (20 calories to the fluid ounce).

The mother was discharged when the infant was getting 561 cubic centimetres (19¾ fluid ounces) of breast milk in the twenty-four hours.

The mother was given a table of the quantities of artificial food to be offered to the baby. If the breast milk increased the infant would not finish its bottle. This was based on the average of the previous five days' breast feeding.

The mother visited weekly to have the infant weighed and the complementary food reduced, if necessary.

During the first week the mother had been able to reduce the quantity of complementary feedings from 284 cubic centimetres to 170 cubic centimetres (ten ounces to six ounces) in the twenty-four hours. The six and nine o'clock feeds were not complemented.

The next week the baby returned, entirely breast-fed six weeks after the first attempt to re-establish the milk was made.

To resume, this baby weighed 3.8 kilograms (eight pounds six ounces) at birth. It was losing weight on strong food having a theoretical caloric value of 412. It was given a diet yielding 120 calories, which was gradually increased. On this food of low caloric value it actually gained in weight, partly, no doubt, due to retention of fluid. As the baby's condition improved, it was able to take over its theoretical amount of calories, thus making up for lost time. As it approaches normal weight, the caloric value of the food taken will approach the average standard.

### The Manual Expression of Milk from the Breasts.

This is the best method to use to make sure that the breasts are thoroughly emptied when the milk has failed and it is desired to increase the supply:

The breast is raised in the hand and gently lifted forward and the area behind the nipple is compressed by the thumb and fingers. The nipple itself is not touched. The motion of lifting and drawing forward the breast is repeated in order to encourage the milk to flow along the ducts into the dilated portions under the areola and the milk is again expressed. These motions are repeated until no more milk can be obtained. Mothers can learn to do this quite skilfully. This is a much more thorough and successful method than using the breast pump.

I show a chart to illustrate how the milk may be increased by artificial stimulation alone in cases where it is impossible for the infant to suck.

To recapitulate the means by which difficulties in breast-feeding may be overcome, the following points should be borne in mind:

(*i.*) That all mothers with the rarest exceptions can suckle their infants either wholly or partially.

(*ii.*) That only under the very rarest conditions does the mother's milk not agree with the infant.

(*iii.*) That if the infant is fretful and not gaining in weight, not sleeping properly, etc., the quantity and not the quality of the milk is probably at fault or there is some mismanagement of the details of the feeding.

(*iv.*) That the milk can be increased by the regular stimulation of the infant sucking at proper intervals, provided that it is certain that the breast is completely emptied at each meal, either by the infant or by manual expression.

(*v.*) That even when the infant has been entirely weaned for some weeks (as long as six weeks in some cases), breast feeding can be re-established by regular stimulation of the breasts by the sucking of the infant, manual expression and artificial stimulation by massage and alternate hot and cold sponging.

(*vi.*) That in the case of premature infants the

breasts can be stimulated to secrete by artificial means until the infant is strong enough to suck.

(*vii.*) That in cases of difficulty, complementary rather than supplementary artificial feeding is required.

## NUTRITIONAL DISEASES OF INFANTS AND THEIR TREATMENT.[1]

BY L. R. PARKER, M.B., CH.M. (SYDNEY),
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SUCH is the title of the subject suggested to me for my paper this evening; but the term is such a wide one, embracing as it does acute gastro-enteritis, acute enteritis not of specific origin, the more chronic forms of enteritis or colitis, including the dysenteric, tuberculous and syphilitic, and finally those types of bowel trouble which have manifested themselves since birth and do not appear to be directly connected with bacterial infections; the term is such a wide one, I say, that naturally it is impossible even to touch upon all it embraces in a space of twenty minutes.

Therefore, it is my purpose to confine these remarks to the last types mentioned, *viz.*, those which have been manifest since birth, and to group them, despite their varied characteristics, under the general title of marasmus or malnutrition.

Before venturing with considerable diffidence to place my observations before you, some of whom were my teachers, while others were versed in this lore before I even dreamt of it, I would like to say that, although much of my matter is drawn from hospital experience at "Greycliff," such experience does not entitle me to approach the subject in any sense from a scientific point of view. Throughout the summer we average from forty to fifty nutritional patients, many acutely ill, and during the remainder of the year we vary from twenty-five to forty, the majority of whom are the essentially chronic and stubborn types. But "Greycliff" merely places at the disposal of its medical officer the above mentioned patients, a fine old house with large rooms, commodious fly-proof verandahs, lovely and adequate outdoor surroundings, an unrivalled harbour site, a reasonably adequate nursing staff, with considerable experience, ability and enthusiasm at the head thereof and a milk supply produced from its own herd. Admirable advantages all of these, but they do not permit one to talk of *post mortem* appearances, bacterial findings, food analyses, etc., etc..

### Symptomatology.

And now to the symptoms and signs of the subject of this paper.

The marasmic infant committed to one's charge may be any age from several weeks to many months, the former entering, the latter generally emerging from the *viâ dolorosa;* but all of them with a history of trouble dating from birth and manifesting a developmental deficiency for their age in every ascertainable direction. The complexion is dull, pallid, opaque; the skin easily spotting and greedy of rashes. Sparse, lustreless hair; usually an ancient but dull expression, capable only of one change, namely, to distress. Extreme emaciation, with every bone showing and every joint disproportionate. The subcutaneous tissues flabby, devoid both of liquid and fat and the muscular development nil. The face is almost invariably the most presentable portion of the anatomy. The abdomen is sometimes sunken, but often protruding, occasionally aldermanic in its proportions, verging on or successfully achieving Hirschsprung's condition. The weight of these infants does not vary with age. With reservations it might almost be said that a child of any age may be any weight. They are often as low as two and a quarter kilograms (five pounds) and are seldom above four and a half or five kilograms (ten or eleven pounds) and, curiously enough, in the matter of prognosis, whereas the weight is of minor, the age is of major importance. In other words, the ultimate chances of a child of three months weighing three kilograms (about seven pounds) are better than those of one of three weeks of the same weight. The former, despite the bad showing, has demonstrated a capacity to live; the latter has still to win his spurs.

But to return to the symptomatology! Last, but not least, the bowel is in trouble, apparently throughout its whole length, for the motions are capable of showing both curd and mucus to varying extent. The numbers vary with the individual and in the same individual from, say, three to seven or eight in twenty-four hours, while the colour ranges from green, greenish-white, greenish-brown, greenish-yellow to yellow. They are always more or less liquid in consistency. Their reaction is only occasionally strongly acid and then only for short periods, so that one is extremely dubious of any of these conditions being associated with purely fat or carbo-hydrate dyspepsias or intolerances. Vomiting may be present or absent, but is exceedingly prone to appear on the slightest provocation. Sometimes it is an exceedingly prominent and persistent feature.

### Causation.

The essential causation must of necessity be obscure, but there can be little doubt that it must be in some way attributed to a delayed glandular development on the part of the child. Not a glandular deficiency in the accepted meaning of that term. The former is a temporary, the latter a permanent condition. As to the initial point of the delay, I am quite unable to surmise. Whether there is a general delay common to all the glands or a delay primarily in the ductless glands, with the digestive glands secondary thereto; or whether in the digestive glands primarily and alone. But for practical purposes I have long adopted the habit of looking upon the condition as due to a delayed development of the digestive glands, with a consequent functional incapacity. Nothing I have yet seen in the way of response to glandular treatment has entitled me to go any farther back than this first link in the possible causation chain.

But if glandular inefficiency be the primary cause of true marasmus, there is a secondary cause and one, too, that is not so veiled in obscurity. Many of

[1] Read at a meeting of the Pædiatric Section of the New South Wales Branch of the British Medical Association on August 18, 1922.

these frail beings have been so battered about by injudicious management at the outset of their careers that, super-imposed upon their original developmental condition, there is a second one of chronic enteritis or intestinal irritability, the two together constituting a problem of the most difficult and tedious nature imaginable. By "battering about" is meant that when in their first days these infants manifest the signs of gastro-intestinal turmoil, it is too frequently and too casually assumed that the initial food (maternal generally, but artificial sometimes when circumstances have demanded it) is the discordant factor. That the child itself may be the originator of the disturbance is not considered until future events proclaim the lamentable fact and, in the case of the maternally fed at any rate, the supply has gone beyond recall. A second medium is tried and perchance a third. A magical substance is being sought for that has no existence—a food that will turn itself into muscle, blood and bone, without the aid of a digestive apparatus with its essential juices. At each successive blow, the child staggers; it loses weight and beauty; the original vomiting and diarrhœa become more pronounced; the child becomes restless and unhappy; hunger, thirst and dyspepsia are bunched together in one indecipherable tangle and the whole picture constitutes that most pathetic of spectacles—a marasmic.

That such battering about can happen to the digestive tract and with deplorable results is evidenced beyond dispute by the scores of cases one sees of chronic enteritis in which the child, after weeks or months of normal progress to vouch for its digestive integrity at birth, has, as the result of some bacterial invasion or dietetic misdemeanour, embarked upon a course strongly akin to that which is the subject of discussion. If, then, a normal bowel can be so hardly hit, how easy it is to appreciate the vulnerability of the immature and undeveloped one and, furthermore, to see that the development which, if unhampered, is probably not so very far behind schedule time, is, under the influence of the new and depressing factor, certain to be retarded to an indefinite degree.

A second reason for one's conviction that "battering" plays a very major part in the production of the true marasmic will (it is hoped) render itself reasonably manifest when, under the heading of treatment, there is discussion of what may happen to certain infants who, one is in the habit of thinking (perhaps with insufficient grounds), would have entered the marasmic handicap but for the fact that the battering was either withheld or at any rate mitigated.

To sum up, then, for the time being let it be taken that the true marasmic is the composite product of a delayed digestive development and an injudicious management at the outset of its career.

If this ætiological statement be correct, it is obvious that the infant's digestive apparatus must be looked upon as a limping or defective machine and, according to all the laws of mechanics, the load that it is asked to carry must not be disproportionate to its carrying capacity. Furthermore, not only is it certain that, if the load be too heavy, a breakdown is inevitable, but also, if it be too light, the vital factor obtrudes itself, development is retarded, the organs deteriorate and, as Dr. Truby King has said, the child may be ingloriously permitted to starve itself to death. How, then, may the load be correctly adjusted or, in plain words, how is the marasmic infant to be fed?

### Prophylaxis.

First of all there is the question of prevention and obviously that can only accrue along the lines of an abstention from battering. This is usually achieved by not withdrawing the child from the breast when at the outset of its feeding career it shows signs of gastro-intestinal disturbance. This statement is trite, I know, but there is still a little more to be said. In each year of one's obstetric practice there are met a certain number of infants who manifest such signs and exhibit a distressing tendency to persist therein. Under such circumstances one of the primary essentials is to lay the salient points of the problem before both parents, emphasizing the impossibility of a point-blank decision in the case of "food *versus* child" and the necessity of a well-considered, persevering test, with the child unfortunately as the only control. Such an understanding is the undoubted due of the parents and when arrived at, generally insures an intelligent and persevering cooperation, which is not only of advantage to the child, but incidentally to the medical practitioner, in that, without it, it is not impossible that his services may be dispensed with before the final achievement and history will then relate that some defter magician was the wielder of the magic wand.

But it is not only necessary to institute a persevering trial of the natural medium. Reverting to the theory of the defective development and the appropriate load, some adjustment seems necessary in a food whose concentration is suitable for a normal digestion. The child's instinct for quantity cannot be left to itself as in the normal infant, for, should it take enough (as at first it will) to satisfy its thirst (not hunger), it will take too much food for its digestive capacity; whereas, if it only be allowed sufficient food, its thirst will go unsatisfied. In either case there is disability. The unfortunate position of a breast-fed infant in time of trouble is that its chop and its glass of water are combined, a fact that should never be lost sight of in other circumstances as well as the present. The appropriate procedure is not obscure and one's own routine is to administer from fifteen to thirty cubic centimetres (a half to one ounce) of water by spoon before each regular feed and to it is generally added 0.12 to 0.18 gramme (two to three grains) of citrate of soda as a digestive adjuvant. If signs of bowel irritability persist—discomfort, wind, restlessness, diarrhœa—small doses of castor oil, say, fifteen to twenty drops once, twice or thrice daily, according to circumstances, often have a marvellously cheering effect.

Under such a régime perserveringly adhered to, one has seen many infants gradually forge ahead and stabilize in a reasonably short space of time and the impression gathered—rightly or wrongly—is that less conservative methods would have rendered

them sorrowful counterparts of many of the "Greycliff" infants.

Before this phase of the subject is left, it cannot be too clearly emphasized that, although it is not a practical measure to estimate the quality of a maternal supply, it is eminently possible to estimate the quantity. Nothing more hopelessly deplorable can happen than that a child diagnosed as a marasmic and presenting—because starved—all the signs of a marasmic, should be kept to breasts which are more or less sterile and empty. The maternal word in this connexion should be viewed askance. I have seen a woman who successfully fed seven children, equally successfully starve the eighth. She did not know the breasts were empty and many women are similarly misled in one way or another. For example, paradoxical though it may sound, empty breasts will not infrequently drop milk at the nipple. The breasts should be examined; the child should be watched while feeding and in case of any doubt weighed both before and after feeds over a period of two or three days. In this connexion a spare weighing machine which can be lent to people who cannot afford their own, is a useful contrivance which will repay in valuable information for any generosity and expense.

Reverting to the question of prophylaxis! In the event of the maternal supply being irretrievably absent or deficient from any cause, then the artificial feeding resorted to should be somewhat on the lines to be laid down presently in connexion with the fully developed case. The general principle is that the fluid should be adequate, the food load light and the child be given an opportunity to adapt itself to it.

### Treatment.

Coming now to the question of the management of a fully developed case of malnutrition, there are obviously three main headings to be considered—dietetic, general and therapeutic. The first two are of the utmost importance, the last of limited value.

*Dietetic.*

With regard to the first (dietetic), for many years past whey has constituted the basis of one's dietetics in these cases and one would like to draw attention to the following analysis of the whey as used at "Greycliff":

| | | |
|---|---|---|
| Fat | 0.75% | One hundred grammes would have a heat value of 24.000 calories as compared with the 66.700 calories of ordinary cows' milk. |
| Sugar | 4.52% | |
| Proteins | 0.73% | |
| Ash | 0.53% | |
| $P_2O_5$ | 0.10% | |
| Total Solids | 6.58% | |

Such whey is not obtained by stripping the fluid from the set junket, but by treating milk as follows: Place cold milk with appropriate amount of rennet over medium gas flame. On reaching 40° C. the milk automatically thickens. Continue the heating process, but somewhat more slowly and always short of boiling, stirring the thick portion meantime over to the side of the utensil with a spoon. This coagulated caseinogen is quickly worked into one solid, dough-like mass, from which all possible liquid should be expressed. The whole is strained and the liquid whey is then seen to be a pleasant, milky-looking fluid, quite different from the thin, creamless material which is obtained from the set junket. Such whey, it will be noticed, has a food value equal to one-third of that of cow's milk. Furthermore, although it contains all the essential food elements, there is a reduced proportion of those (the fats and proteins) most likely to impose strain on an impaired digestion. Moreover, the proteins are of the most digestible type, such an absence of the more resistent caseinogen not being obtainable in any other dilute form of suitable infant food. Such whey is exceedingly palatable; all infants without exception delight in it and this palatability induces them to take their full quota of liquid, a most vital point in the cases under discussion.

From a practical point of view, whey will for a period increase rapidly the weight of most wasted infants; obviously those who have lost tissue fluid in vomiting or diarrhœa or have been starved of liquid in the attempt to starve them of food. It will also maintain the weight of any infant for a prolonged period when administered in the same quantity or very little more as the same infant would have taken of its ordinary food in ordinary health. For instance, I have seen a child of 8.2 kilograms (eighteen pounds) maintain its weight and reasonable condition for three weeks on the same quantity of whey as it would ordinarily have taken of its milk mixture. Hundreds of frailer children are seen to do the same for longer or shorter periods. One child seen in consultation recently, literally starved from over-feeding, promptly put on five hundred grammes (one pound) in a week on whey, much to the consternation of the physician in charge, who, apprehensive of an operation for pyloric stenosis, feared that the child would go still further downhill on such a "weak" food as whey.

For its nutritive and digestive qualities, its palatability and its fluid value, one has come to depend on whey as an almost vital adjunct in the treatment of all malnutritional conditions. And unless there is some obvious reason to the contrary, all such infants entrusted to one's care are submitted to the whey test in the full knowledge that, while it is being applied, they will neither be disastrously starved nor egregiously over-fed. By systematically using whey at the inception as both food and test, one is able to form a reasonably accurate estimate as to the intrinsic severity of the intestinal irritability or enteritis. Such a test is essential in hospital, for the bowel signs on admission are not necessarily any criterion of the actual bowel condition.

Presuming then that an infant has demonstrated its capacity to advance by a satisfactory response to the whey, either as a test or as a diet administered for a longer or shorter period, the question of the introduction of a more concentrated medium naturally arises. In this connexion, although one usually handles either condensed milk, cows' milk, "Lactogen" or "Benger's Food," experience consistently indicates that it matters considerably less what you use than how you use it. Condensed milk, because of its well-known characteristics, still maintains a very foremost place in the feeding of un-

stable infants and in hospital, where its weakest point (excess sugar) is neutralized by the combination of equal parts of the sweetened and unsweetened brands, it is most useful. Outside hospital this combination is usually too expensive to be practicable, as the unsweetened brands are unstable and not to be relied on for more than twelve to twenty-four hours after opening. "Lactogen" is an excellent food under most circumstances. Cows' milk, as supplied in Sydney, is essentially risky and for that reason "Greycliff" infants are not accustomed to its use before their discharge as was formerly the practice. Most of our supply is used for whey. "Benger's Food" is an extraordinarily good food, generally used for older children; but it can be given to young infants with excellent results.

Whichever medium be selected, the bowel manifestations, weight chart and, most important, the report as to the desire for food on the child's part being used as guides, an advance is made on the following lines, *viz.*, that a certain amount of the new medium at standard strength is substituted for the same amount of whey in the original whey feed. This amount varies naturally with the capacity of the child. It may be fifteen cubic centimetres (half an ounce) or less in an obviously unstable or very young child; it may be sixty cubic centimetres (two ounces) or more if the indications are exceedingly favourable; but, under any circumstances, seeing that it is a substitution and not an addition, the change is a more gradual one than is at first sight apparent. From then onwards a series of such advances made at varying intervals, punctuated perhaps by various long stationary periods or retreats, according to the response of the child, becomes the order of the day. Advances are indicated by an improvement in the motions and general appearance and particularly by a manifest desire on the part of the child for its food. Indications for a retreat are increasing pallor and loss of appearance, unfinished bottles, vomiting, disturbed motions, a rapid drop in weight. The latter is a dramatic symptom viewed on a chart and, although it not infrequently occurs without any cause ascertainable at the moment, it always heralds trouble within a day or so. Such trouble is not always directly nutritional in character, but may be broncho-pneumonia, pyelitis or some other intercurrent affection. In any case, however, a dietetic retreat is generally indicated.

The rate for advancing cannot be worked out on any rule-of-thumb method; but it is better to err on the side of slowness. Under favourable circumstances an increase is seldom advisable under five or six days. The child must be given ample time to manifest its attitude towards the increase before a further change is decided upon.

In spite of the greatest care it is impossible always to read the signs aright and it is certain that some are kept back who might have been pushed on more rapidly, while others, unfortunately, are pushed back by premature advance. Others again will fall back for no obvious reason whatsoever. In the case of relapse it is seldom necessary to consider a change of concentrated medium. One has seen many scores of infants who, having been tried unsuccessfully with various foods, have eventually sailed right out of their charts on one which apparently achieved nothing, or worse, on a previous occasion. Indeed, throughout the management of these infants there is a dominant feeling in one's mind that, although everything that can be done with the food, must be done, a time is being waited for and, until it comes, full success is impossible. That such time heralds the more or less completion of the developmental processes there is little doubt in one's mind and the problem that presents itself in the mean time is that of keeping the child alive and ministering to its development, by giving all that can be carried, but without overloading.

The period of management extends over months and during the greater part of it, despite a perhaps considerable total increase in food administered, there is an extraordinarily slow increase in weight. The most notable change for the better is in the appearance and one can only describe it as an appearance of increasing stability.

One of the final phenomena in long-standing cases is extraordinarily interesting and is always watched for with a certain amount of eagerness. It is that over a period of some weeks during which there may be little or no increase in weight, the skin texture undergoes a noticeable change and becomes visibly and increasingly transparent, the transparency being followed by the gradual appearance of a colour. At or about this stage—an interesting point—often without any increase of food, the weight will begin to rise steadily and, moreover, will continue to rise until the poor little bag of skin and bone eventually becomes a real live baby.

The question might well be asked of a practitioner who, like myself, has a conservative idea on the subject of infant feeding, whether many of these long-drawn-out cases may not have responded to bolder and more vigorous methods. One has often asked oneself the same question and expects to ask it again and again; and, naturally, a practical answer has been sought for in what appeared to be suitable cases. Children have been selected in whom the bowel has become reasonably stable, but who, although their appetite demand has apparently been satisfied, yet are making no weight gain. These have had their food progressively increased at a more rapid rate than usual. Very rarely one may forge ahead. Among the remainder there are three types of response. Some will at once show gastro-intestinal disturbance; the others will show no such disturbance, but will either maintain a stationary weight or will slowly begin to lose it. Another most convincing proof that extra food is not the solution of this conundrum is the common spectacle of two children, perhaps side by side, the one small, but, though heavily fed, making no advance, the other almost twice the weight, but advancing rapidly on almost half the quantity of its diminutive neighbour.

Time does not permit me to do more than touch upon this feeding question; but one point must not be omitted. There must be no attempt to feed these children on any caloric theory or with any idea that because they are of certain weight they should only require a certain bulk. Unvarying experience shows that they must have their stomach capacity satisfied and, moreover, that that stomach capacity seems almost invariably proportionate to the age of the

child and in no way proportionate to the child's own bulk. One often stands aghast at the sight of some whipper-snapper of a "seven-pounder" imbibing its one hundred and eighty cubic centimetres (six ounces) or more of liquid and essays to reduce the inebriate's allowance. The protests are immediate, vigorous and unmistakable and one resigns oneself more readily in that, although many of these infants report themselves afterwards, in none of them can there be detected by symptom or sign the dilated stomach said to be achieved by excess food bulk. It can only be assumed that the more dilute food is absorbed so rapidly that the stomach distension is of too short duration to do much damage. Thirty cubic centimetres (one ounce) or slightly more of food for each month of life up to eight months at three-hourly intervals is a moderately accurate guide as to quantity.

In concluding these remarks on feeding, one might add that where extra carbo-hydrates are indicated, coffee or arrowroot biscuit pulp is very suitable, while a fat deficiency, as in the condensed milk mixtures, is supplied by butter, which, besides being safe and for this purpose at any rate economical, mixes excellently with the warm fluid and is obviously the nearest approach to cream.

*Therapeutic Measures.*

With regard to these I have little to say. Bismuth and opium are seldom used. The bowel should be kept in order with dietetic measures, together with small doses of oil in times of trouble. Citrate of soda is useful, while mercurial indications are best satisfied by inunctions.

As to glandular substance administrations, I can say but little at present. Even if one's own beliefs were not in the direction before indicated, ignorance would have to be admitted unreservedly as to which was likely to be the defaulting gland in a young infant and this despite the fact of a most careful perusal of the list of symptoms said to be associated with such deficiencies. On a number of occasions, feeling that pituitrin, being a stimulant, might at any rate do little harm, I have administered small doses, but have never seen any encouraging results. Recently "Hormotone" has been tried as an all-embracing remedy and certainly some infants have done well at the time of administration. But I am not yet satisfied that such time did not coincide with other events. The new Commonwealth serum for dysentery has not yet been used at "Greycliff," but it is more likely to be useful in cases of acquired enteritis than in those under discussion.

*General Treatment.*

The general management of these infants is exceedingly important. Warm or hot baths should always be administered, even to the most fragile, for, not only is the maintenance of cleanliness vital, but it is impossible to see these infants after their bath without recognizing that they are stimulated by it to an extraordinary degree. Light clock-wise abdominal massage is most useful in case of bowel dilatation and a reasonable amount of steady nursing is part of the necessary régime, especially in the older ones; but it must be well away from meal times, as many of these children vomit on the slightest provocation. In hospital a deficiency of nursing is best compensated for by propping even young infants into a sitting posture from time to time.

Clothing is a vital matter, but not always in the direction of putting it on. In the winter they should be kept exceedingly warm with body clothing and bedding; but in hot, summer weather it is of paramount importance that they should be stripped to a considerable degree. To sweat one of these infants—and they do sweat with a vengeance—under heavy blankets or under-clothing is a serious error which, if persisted in, becomes criminal. Most of our text-books on infants are written in England, where the depressing effects of cold are an important factor, but in our part of Australia at least I am convinced that of the two factors, heat and cold, the former is the greater enemy to children. Extremes of either are unfavourable and this point should govern the question of outdoor on any given day. Outdoor under moderate conditions is excellent for children who are progressing favourably; but those who are ill are best kept under more sheltered conditions.

## OBSERVATIONS ON THE MODE OF DEVELOPMENT OF BROOD CAPSULES AND SCOLICES IN THE ENCYSTED STAGE OF TÆNIA ECHINOCOCCUS.

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### Introduction.

The original research of Leuckart[1] in 1862 has remained for many years the main source of information as to the development of the encysted form of the *Tænia echinococcus*. His description was based on observations made on the cystic stage in the artificially infected pig and dealt mainly with the later stages of the development. He approached the subject mainly from the biological aspect and was influenced a great deal by the analogous development of the other cestodes. His description is in the main accurate, but presents some inaccuracies and is at times rather confusing. With modern technique, serial sections and the like, Dévé[2] has filled in an important gap in our knowledge of the histogenesis of this interesting parasite. He has followed the minute details of the development from the earliest invasion of the liver by the six hooked embryo at about the third hour after infestation to some five months later, when a definite two-layered cyst with well-marked adventitia is present.

The exact mode of development of the brood capsules, the scolices and daughter cysts has always been somewhat uncertain and confusing. I have recently completed an investigation of the first part of the problem.

### Material and Methods.

The hydatid material was at first obtained from cysts of the liver and lungs of the sheep. These were obtainable in practically unlimited quantities from the abattoirs and a weekly supply was brought to the Institute for the preparation of the specific

hydatid antigen for the complement deviation test for this disease.[3]

During the last year hydatid cysts removed at operation from patients in hospital have also been studied and it is in these specimens that most of the important early stages have been seen.

The specimens of cysts, brood capsules and scolices have all been examined in the fresh state, stained and in serial sections. Altogether hundreds of cysts have been examined and sectioned.

### Development of Brood Capsules.

The parasite, as usually observed, consists of an external laminated hyaline protective layer, which is lined on its inner side by the active parenchyma or nucleated layer which in turn surrounds the specific fluid.

The nucleated layer is very thin, sparsely supplied with cells, rich in glycogen and here and there shows calcareous particles. It can usually be separated from the outer layer without much difficulty. From this layer are developed brood capsules, daughter cysts and the laminated outer layer.

The brood capsules first make their appearance as a multiplication of one of the small nuclear masses of the parenchyma, just showing as a small elevation on section as yet solid. Its wall rapidly thins and it becomes vacuolated, forming a small vesicle. This becomes pedunculated and in the normal state remains attached to the parenchyma by a very delicate pedicle. Trauma, however, readily sets them free and unless the cyst is carefully handled, they are found floating in the cyst fluid. They can usually be seen as small white particles shining through the cyst wall and may be present in thousands. They do not develope evenly over the whole of the parenchymatous area, but tend to be aggregated in particular spots.

I must join issue here with many of the previous observers and maintain that, except perhaps in the very late stages, there is no differentiation of the thin brood capsule wall into two layers.

From the brood capsules the scolices or future worm heads develope (see Figures I., II., III., IV., V. and VI.).

### The Scolex.

The first sign of the scolex is a local thickening of the wall of the brood capsule and this rapidly developes into a small bud on the inner surface of which hyaline cuticular material is laid down.

The wall here tends to evaginate, as an external cupping of the wall, and the papilla sinks into this and developes further in this evagination (see Figures VII., VIII., IX. and X.).

This external cupping of the wall may be due to the growth of the brood capsule cavity not keeping pace with the rapid development of the growing scolices and the giving way of the wall at the softer and more cellular area. Probably, however, it is simply a protective mechanism to guard the delicate developing head.

The condition shown semi-diagrammatically in Figure X. is a very common one, especially in the cases where the brood capsule is already packed with more or less mature scolices.

The head, even at this stage, contains contractile cells, though there is little or no differentiation of structure and soon by the active contraction of these cells the head is forced into the cavity of the brood capsule (see Figures XI. and XII.).

Once inside the brood capsule it developes rapidly, the typical shape appears, the stalk becomes marked off, the anterior cells become differentiated and the form of the suckers appears. The posterior or body cells become elongated and arranged in longitudinal strands, while the hyaline cuticle is well marked and spreads on to the stalk. The hyaline material near the head end becomes thickened and here a circlet of hooklets is rapidly developed (see Figure XIII.).

This differentiation goes on until the main features of the mature scolex are apparent, while calcareous bodies make their appearance in the body. The scolex, alone or in company with others of its kind, remains attached to the brood capsule by a fine stalk and swings freely in the brood capsule. In order that the important hooklets can develope safely and to keep them from becoming knocked off by the active movement of other scolices, the contractile body of the scolex acquires the faculty of invaginating the head.

Thus the typical resting position of the scolices is assumed, with the hooklets inside, their free extremities pointing anteriorly (see Figures XIV. and XV.).

The hooklets are always reversed when turning in, so that they will not catch on the inverting suckers.

When the reverse action takes place and the head is protruded, the hooklets are first put straight and follow, so that they do not catch (see Figure XVI.).

This is the typical and common mode of development, but there are several variations.

In the case of the first few scolices developing, the evagination or external cupping stage, as represented in Figures IX. and X., is not a marked feature, though an indication of it is seen in Figure V.. This is probably due to the fact that protection to the delicate growing head is not required, as there is nothing in the brood capsule to affect it.

Hence in the very young capsules straight forward internal budding is the rule.

The scolex, too, has the power of in-and-out movement as regards the evagination for some time, until the development of a constriction at the region of the future stalk precludes this.

At times the development and differentiation of the scolex, even to the formation of hooklets, occurs in the evaginated bud. This is merely a prolongation of the time taken over the stage represented in Figure X..

Following this typical development, the scolices grow from the wall of the brood capsule and may be seen in all stages of maturity, while up to forty scolices may be seen in a capsule.

Each of these scolices has a definite hyaline cuticle which runs on to the stalk and on to the neighbouring part of the inner wall of the capsule. In this way the inner wall of the brood capsule appears to have a partial hyaline layer.

This layer is never complete, is never a distinct feature and is only seen in old, well-matured capsules.

A great deal of stress has been laid on this reversal of the layers in the brood capsule. This is

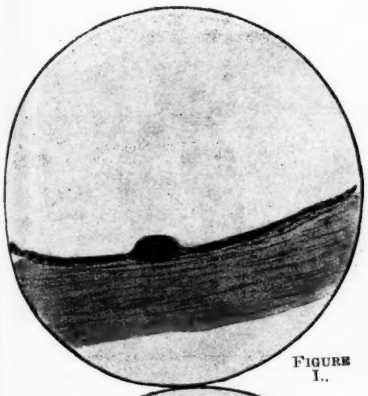

FIGURE I..

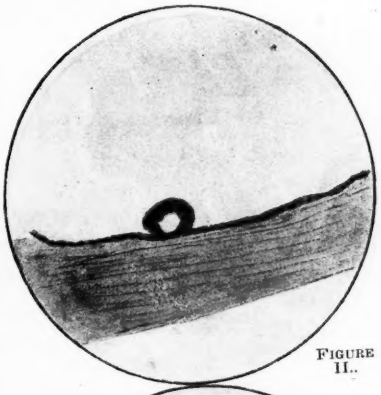

FIGURE II..

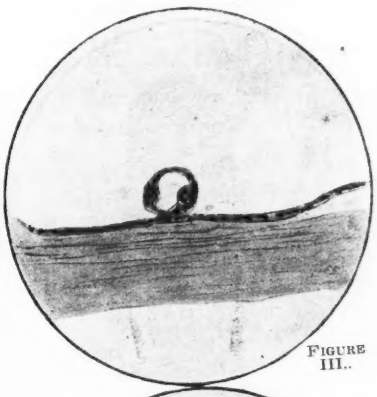

FIGURE III..

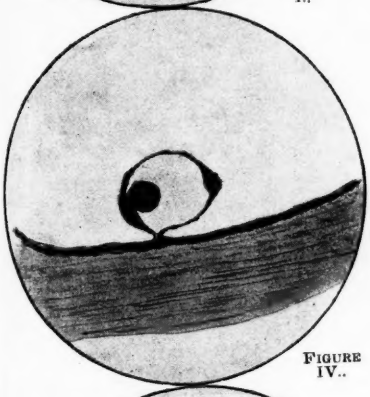

FIGURE IV..

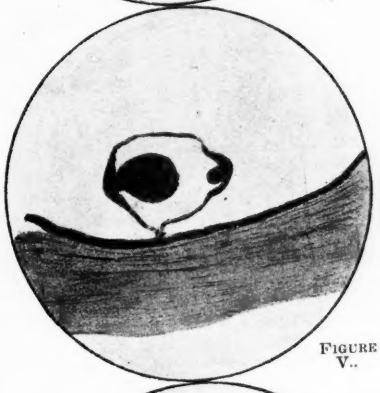

FIGURE V..

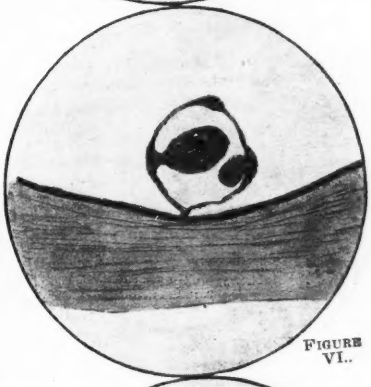

FIGURE VI..

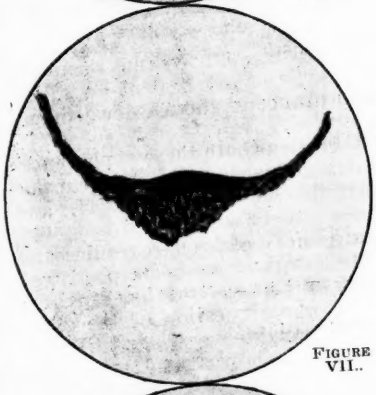

FIGURE VII..

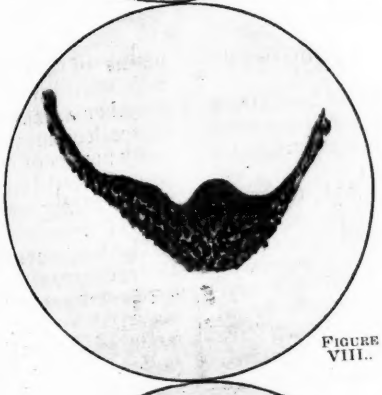

FIGURE VIII..

FIGURE IX..

FIGURE X..

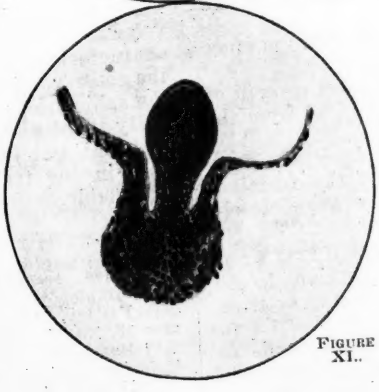

FIGURE XI..

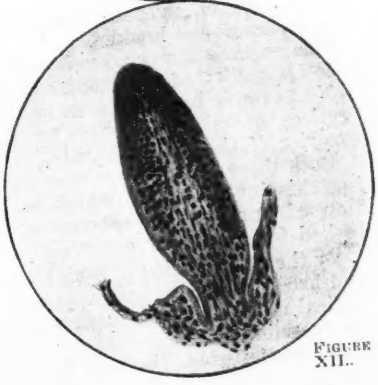

FIGURE XII..

FIGURE XIII..

FIGURE XIV..

FIGURE XV..

FIGURE XVI..

FIGURE XVII..

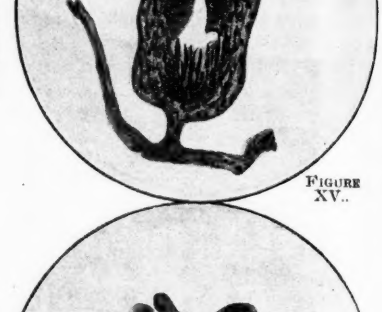

FIGURE XVIII..

unnecessary, as it has nothing to do with the development of the scolices.

The scolices are actively contractile and on stimulation by warmth protrude their heads and impart a certain degree of pseudo-peristaltic movement to the brood capsule.

In contra-distinction to Leuckart and others, I find no evidence of a vascular system in the brood capsules or scolices; their nutrition and indeed that of the whole cyst are dependent on osmotic processes.

Not all the scolices reach maturity and it is common to find among the mature members of a brood capsule detached degenerate scolices which are characterized by their dark brown colour, shrunken bodies, lack of glycogen and calcareous bodies and poorly developed hooklets.

The scolices always develope in the above manner into the cavity of the brood capsule.

No example has been seen of the direct growth of scolices or stalks of scolices or of budding from the exterior of the brood capsule.

Both these methods are commonly depicted. During this investigation, which covered the examination of hundreds of specimens, they were closely looked for but never observed.

By the examination of serial sections of cyst walls and brood capsules these apparent exceptions to the typical mode of development were proved to be traumatic artefacts.

When ruptured, the thin brood capsule tends to turn inside out, so that the picture of external budding of scolices may be readily simulated. The remains of the brood capsule, flimsy and shrunken, may surround the pedicle of attachment or adhere together and the appearance of stalked development of scolices may be copied.

By means of serial sections both these appearances were proved to be artificial, the break in the capsule being always detected (see Figures XVII. and XVIII.).

The "degeneration" (*sic!*) of brook capsules and their conversion into daughter cysts as portrayed by Rasmussen,[4] Nauyn[5] and others has not been observed as yet. The whole question of the exact mode of formation of daughter cysts is still *sub judice*. Many interesting observations have been made and are being further investigated. This development of daughter cysts appears to be almost a characteristic of the disease in human beings and the consideration of this problem will, I hope, form the subject of a future communication.

My thanks are due to all those who have sent me material and especially to Mrs. E. Barnes, whose help in the technical side of the work has been invaluable.

**References.**

(1) Leuckart: *"Die Parasiten des Menschen,"* Leipzig, 1886.

(2) Dévé: *Archives de Médécine Experimental et d'Anatomie Pathologique, Tome* 27, No. 2, *Mai*, 1916.

(3) Fairley, N. H.: *Quarterly Journal of Medicine*, Volume 15, No. 59, April, 1922.

(4) Rasmussen: Translation by F. von Mueller, *The Australasian Medical Gazette*, 1869.

(5) Nauyn: *Archiv für Anatomie und Physiologie*, 1862, *Seite* 612 *et sequentiæ*.

# PRELIMINARY REPORT ON THE CAUSAL ORGANISM OF BLASTOMYCOTIC MENINGITIS IN AUSTRALIA.

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In 1917 Swift and Bull,[1] of Adelaide, reported the first case of blastomycotic meningitis recorded in Australia. Following this Barlow[2] reported a similar case in the same city in June of the present year, while in August, Williams[3] wrote in some detail on the pathology of a fatal case in Melbourne.

I have the permission of the Director-General of Public Health of New South Wales to mention that a patient died in the Coast Hospital, Sydney, in July of this year from the same disease. I was asked by Dr. E. W. Ferguson, Principal Micro-Biologist of the Department of Public Health, to determine the systematic position of the organism found *ante mortem* in the cerebro-spinal fluid and *post mortem* in the meninges of this patient. My thanks are due to Dr. Ferguson for the material and his ever-ready assistance during my investigation.

Though I have not examined the cultures from the cases in Melbourne and Adelaide, still I have little doubt from the morphology of the organism described, its cultural characters and the results of animal inoculations that it is identical with the organism isolated from the Sydney case.

So far the organism has not been identified. The report of Swift and Bull[1] drew attention to the gelatinous capsule and noted that the figure in Coupin's "Atlas des Champignons" of *Saccharomyces tumefaciens* showed such a capsule.

Their primary cultures made on unsuitable media were poor, but through this they were able to detect hyphæ of rudimentary types. They described highly refractile endospores, but it is obvious that these were only the refractive droplets which these organisms produce in adverse circumstances. Rabbit and guinea-pig inoculations made by them were resultless. They concluded that the organism appeared to be that commonly designated as *Cryptococcus gilchristi*, but an examination of the literature would have revealed the fact that the organism causing Gilchrist's mycosis possessed different characters, readily forming hyphæ in culture, as described and figured by Hamburger[4] and other writers.

Barlow[2] demonstrated a yeast-like organism from the cerebro-spinal fluid of a child suffering from chronic meningitis and considered it to be identical or closely similar to *Cryptococcus gilchristi*.

Williams,[3] not finding hyphæ in cultures or in inoculated animals, left the parasite unnamed.

In the course of my investigations during the past two months I have been fortunate enough to obtain by appropriate methods sufficient information to show that the organism isolated from the Sydney case of blastomycotic meningitis is a true pathogenic yeast. I shall describe it for the present as *Saccharomyces tumefaciens* (Curtis), believing that certain new facts which I have ascertained, may yet be found to refer also to this yeast and that the formation of a new species for the Australian form may not be warranted. I have observed the formation of hyphæ corresponding to the forms described for *Saccharomyces tumefaciens*, but better developed, and of asci containing from one to four ascospores in each ascus and have seen the germination of the ascospores. The biochemical reactions of the Australian form correspond closely with those of *Saccharomyces tumefaciens*, as reported by Curtis[5] and Buschke.[6]

In regard to the pathogenicity of this yeast to animals, my experiments are not yet completed, but so far they agree with those done with *Saccharomyces tumefaciens*.

Since Curtis[5] reported the original case in which this pathogenic yeast was found, not more than three or four cases have been recorded.

No doubt many cases of infection by this organism have been wrongly ascribed to *Cryptococcus gilchristi* (synonym, *Blastomyces dermatitis, Mycoderma dermatitis*), which causes many cases of cutaneous blastomycosis, especially in Chicago.

Stoddart and Cutler,[7] of Boston, described in 1916 six cases of blastomycotic infection in which the meningeal lesions were marked, as due to a yeast-like organism which they called *Torula histolytica*.

The pathology of their cases bears a striking resemblance to that seen in infection by *Saccharomyces tumefaciens*, but they were unable to induce sporulation or hypha formation. Their notes on the biochemical reactions are scant and their work has been criticized by Pinoy[8] among others.

In view of the occurrence in Australia of several fatal cases of blastomycotic meningitis in the last few years it may be considered that possibly some obscure cases of chronic meningitis may be due to this infection.

As my investigations on this organism may not be completed for some months, I have considered it fitting to publish this note.

## Literature.

(1) Swift and Bull: "Notes on a Case of Systemic Blastomycosis: Blastomycotic Cerebro-Spinal Meningitis," The Medical Journal of Australia, September 29, 1917, pages 265 to 267.

(2) Barlow, D. L.: "Cryptococcus Gilchristi," The Medical Journal of Australia, June 24, 1922, page 700.

(3) Williams, J. R.: "A Case of Systemic Blastomycosis," The Medical Journal of Australia, August 12, 1922, pages 185 to 189.

(4) Hamburger, W. W.: "A Comparative Study of Four Strains of Organisms Isolated from Four Cases of Generalized Blastomycosis," *Journal of Infectious Diseases*, 1907, Volume IV., page 201.

(5) Curtis, F.: "*Contribution a l'Étude de la Saccharomycose Humaine*," *Annales de l'Institut Pasteur, Tome* X., 1896, page 481.

(6) Buschke, A.: In Kolle and Wassermann's "*Handbuch der Pathogenen Mikroorganismen*," 1913, *Band* V., *Seite* 155.

(7) Stoddard and Cutler: "Torula Infection in Man," *Studies from the Rockefeller Institute*, Volume XXV., pages 1 to 98.

(8) Pinoy, E.: *Bulletin de l'Institut Pasteur, Tome* XIV., 1916, page 401.

# Reports of Cases.

## MALIGNANT PUSTULE OF FACE.[1]

BY R. J. MILLARD, C.M.G., C.B.E., M.B., CH.M. (SYD.), D.P.H. (CAMBRIDGE),

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THE following three cases of anthrax are reported as being of general interest:

I. W. K., male, aged thirty-one years, employed at the railway shunting yards, was admitted to hospital on August 21, 1922, with a history of having scratched his face three days previously and having subsequently used a new shaving brush. A pimple developed at the site of the scratch and the right cheek became swollen.

On admission to hospital a typical malignant pustule of the face was present. The centre was dark and surrounded by a vesicular area, the whole being about the size of half a crown. The face and neck were much swollen with brawny œdema. Slides prepared from the vesicular fluid revealed numerous anthrax-like bacilli.

Under general anæsthesia the pustule and about 1.25 centimetres (half an inch) of surrounding skin were excised. The raw surface was swabbed with tincture of iodine. At the same time an intravenous injection of seventy-five cubic centimetres of anti-anthrax serum was given.

By the next day the œdema had subsided to a considerable extent and recovery was uneventful.

Laboratory reports confirmed the diagnosis. A guinea-pig inoculated from the pustule died in two days of anthrax. The shaving brush used by the patient was also tested; washings of the hairs imbedded in the handle were injected into a guinea-pig and it died of anthrax. Another guinea-pig also inoculated from a similar shaving brush, bought from the same shop, died of anthrax.

II. D.L., male, aged twenty-two years, milk vendor, was admitted to the Coast Hospital on August 23, 1922, with a history of having been shaved by a barber on the morning of the eighteenth and of having developed a sore on the cheek two days later. On August 22 he went to the Royal South Sydney Hospital, where anthrax was diagnosed.

On admission to the Coast Hospital a typical malignant pustule was present on the left cheek. Its size was slightly larger than a shilling piece and it was accompanied by much œdema of the face and neck, extending on to the chest. The patient's temperature was 38.3° C. (101° F.).

Under general anæsthesia the pustule was excised, as in the previous case, and the raw area swabbed with tincture of iodine. One hundred cubic centimetres of anti-anthrax serum were injected intravenously. The specimen has been preserved and is now exhibited.

On the day following the operation the patient was much better and the œdema was subsiding. Progress since then has been uninterrupted.

III. C.S., male, aged twenty-one years, a storeman, was admitted to hospital on September 7, 1922, with a history of having cut a pimple while shaving on September 1, 1922. On September 4 he noticed a hard pimple on the cheek and, thinking it was a boil, he poulticed it. He went to the Royal Prince Alfred Hospital three days later. Anthrax bacilli were found at that institution and he was transferred to this hospital.

His temperature on admission was 37.8° C. (100° F.). He was complaining of headache, but had no other symptoms. One hundred cubic centimetres of anti-anthrax serum were given intravenously. The pustule has an area of about two and a half centimetres in diameter. Its surface is vesicular and it is surrounded by a moderate amount of brawny œdema. A slight improvement has taken place, but it may yet be necessary to excise the pustule.

The patient's shaving brush is of the same make as that of patient W.K.. It is now being examined for evidence of anthrax infection.

In this journal of August 20, 1920, I published notes of seven cases of anthrax which had been treated at this hospital between February and August, 1920. Since then there have been treated here in 1921 three cases and in 1922 one in January in addition to the three now described.

Of these fourteen patients nine had the "malignant pustule" on the face. In the case of at least five of the nine, the shaving brush used was proved to be infected with anthrax. All the patients have been treated with excision of the pustule and intravenous injection of anti-anthrax serum, except the last, who, as above described, has been treated with serum alone. All have been discharged cured.

*Post scriptum* (September 19, 1922).—Excision was not done, as the œdema rapidly subsided and the pustule dried up. The patient is being discharged to-day.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on September 8, 1922.

# Reviews.

## PSYCHO-ANALYSIS.

FOR those who wish to arrive at some understanding of what is meant by psycho-analysis and of the methods used by the adherents of Freud in probing into the mysteries of "the unconscious," Dr. H. Somerville has written an introductory handbook[1] which sets out in a simple form the contentions of the psycho-analyst in relation to "unconscious mentation."

According to the psycho-analyst there exist repressed groups of associated ideas with their accompanying emotional tone, which are the potential cause of dreams, psycho-neuroses and hallucinations. They are represented in consciousness in distorted and disguised form, so that their recognition and interpretation require considerable psycho-analytic skill.

The author describes the methods by which various complexes are brought to light; throughout the book his confidence in the value of psycho-analysis as a therapeutic measure and in the influence of certain complexes, supposed to have an infantile origin, is manifest. In a small compass he has made clear the meanings attached to such terms as "libido," "sublimation," "transference," "fixation," "displacement," "narcissism" and so forth, each of which has for the psycho-analyst a highly specialized significance.

Ingenious arguments are used to show how such deeply buried complexes as the Œdipus, homo-sexual and anal-erotic complexes are represented in consciousness. These complexes are apparently held to be universal in mankind and auto-erotism and homo-sexuality to be merely stages in the process of sexual evolution.

There are numerous illustrative cases given in the book. An example may be given to show the depths to which "the unconscious" may be probed by psycho-analysis. The patient dreams that he is drowning and becomes stuck in a narrow passage through which water is running. "The analysis left no doubt," says the author, "but that it was a birth phantasy"—pre-natal mentation!

It requires no little courage to accept some of the arguments of the psycho-analysts; but without recommending their acceptance it may be said that the author has given in an abridged form a clear presentation of the practical application of the psycho-analytic method.

[1] "Practical Psycho-Analysis," by H. Somerville, B.Sc., F.C.S., L.R.C.P., M.R.C.S.; 1922. London: Baillière, Tindall & Cox; Demy 8vo., pp. 142. Price: 6s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 30, 1922.

## Neo-Natal Physiology.

IN this issue we publish an account of a discussion on the nutritional disorders of infancy. Few subjects call for more careful or more serious consideration and few subjects are habitually handled in a more unscientific fashion. For this reason, if for no other, the thoughtful contribution by Dr. Margaret Harper and the ingenious, if somewhat speculative, paper by Dr. L. R. Parker will be read with interest. Several speakers maintained that the teaching given at our medical schools to medical students in regard to the feeding of infants is inadequate and unsound. It might with justice be asserted that in all parts of the world the teaching is unsatisfactory, largely because the exact nature of the metabolic processes associated with some of the disorders of early infancy have still to be determined. In the Bertillon classification scheme there appears the ill-defined group of "marasmus, debility, asthenia." Corresponding to these vague terms there exists in the minds of the majority of clinicians a hazy conception of some disturbance of digestion in early infancy which is usually treated by some arbitrary method, based on mass impressions. Progress may be attained laboriously and irregularly by these empirical means, but the only safe method of attacking the problem of the prevention and cure of these disorders is by a planned study of the chemistry and chemico-physics of infants of the type included under the unfortunate term marasmics. The study has to be started in its ante-natal stage, for there is indication that the predisposition to the metabolic disturbances at all events is laid down some time before birth. Physiologists have endeavoured to trace the genesis of the power of digestion in the fœtus and they have been successful to a considerable extent. Their investigations, however, have been limited to the normal fœtus and hitherto no elaborate observations have been conducted with a view to the distinction between the several types of fœtus. Dr. Parker's hypothesis of a glandular defect is capable of being tested. An attempt should be made to ascertain whether structural and functional defects in the thymus, thyreoid, para-thyreoid, pituitary, pineal, supra-renal and sexual glands during their evolutional stages correspond with the characters of the infant in whom nutritional disorders are prone to occur. While it would seem to be hazardous to postulate a disturbance of these glands in the marasmic infant, it would certainly be unjustifiable to deny the possibility of such a basis before the closest study has been carried out into the histology and bio-chemistry of these glands in the fœtus before birth and in the newly-born infant. The suggestion has been made that the digestive secretions are dependent for their physiological activity on substances analogous to immune bodies or antibodies. It is known that the introduction into an animal organism of any organized chemical substance results in the appearance of a ferment-like activity usually spoken of as an antibody. Experience has shown that the proteolytic ferments, the ferments capable of converting starch into sugar and the enzymes capable of splitting fats and of hydrolyzing them appear in the later stages of intra-uterine development. At first the enzymic action is relatively limited and unstable. The newborn babe can readily be educated to convert foreign proteins into peptones and to hydrolyse foreign fats, but during the process of training the delicate organs of digestion are apt to revolt. If the assumption that the enzymes appear in the digestive glands in response to the absorption of antigens supplied through the maternal circulation is correct, it would be easy to explain why the fats, proteins and sugars provided in the mother's milk are the best suited to the infant's digestion in the first weeks of life. It would need no further premiss to explain the inability of the infant to deal with large quantities of this, the most perfect, food. A healthy, well-developed infant usually overcomes the difficulty imposed upon it if its half-developed powers of digesting foreign proteins, fats and starches are exercised. The infant can be taught within a very short time to digest starch and to metabolize it. Unfortunately no one has yet demonstrated the

essential differences between the normal infant that can be trained in this manner to deal with foreign proteins, fats and starch and the abnormal infant whose metabolic powers break down as soon as an extra load is imposed on it. Clinical observation would show that there must be several unrelated differences. It is the task of physiologists to lend their aid in elucidating these intricate problems and in establishing a proper basis for the pathology of the various forms of nutritional disturbances of early infancy.

In addition to the predisposing causes of these disorders, there are obviously several exciting factors. Dr. Margaret Harper has exhibited wisdom in recognizing the physiological defect which leaves the infant with too narrow a margin to justify an early training of the function to digest foreign foods. Her insistence on the use of the scales in the feeding of infants and on measuring the infant's needs in terms of calories must be commended. Clinicians are apt to overlook the fact that the heat supplied by food taken by infants is required to provide not only for the ordinary heat loss through the skin and for muscular activity, but also for the process of growth. Moreover, there is a varying wastage of calories of not less than 10% in connexion with undigested food. For this reason it is of great importance to supply a considerable excess of calories beyond the actual metabolic requirements. It is regrettable that the Professor of Medicine of the University of Sydney should have expressed his disapproval of this doctrine, for it is founded on accurate knowledge and cannot be replaced by any haphazard measure with safety. Dr. Harper might have gone one step further. She might have insisted on the control by a chemical analysis of the urine and fæces. There are means at the disposal of the clinician to ascertain with certainty whether the foods, as proteins, fats, carbohydrates and salts, are being properly metabolized. More could be learned by the study in the respiration calorimeter of the manner in which abnormal infants under varying conditions deal with these food ingredients. Dr. Selwyn Harrison described a set of processes which take place in some infants during the course of nutritional disorders. A full appreciation of the limitations of the power of the digestive glands of the infant is essential for the understanding of the exciting causes of these disturbances. But the factors mentioned do not exhaust the list. It has been shown that the thermic balance in the infant is extremely delicate and is readily lost. The importance of the prevention of an adequate loss of heat liberated by the metabolism of the food ingested must be recognized. Clinicians know that babies respond quickly to food injuries by fever and that the febrile infant is liable to invasion by bacteria. The febrile baby seems to lose its power to metabolize fats before its protein assimilation suffers. To feed an infants whose thermic regulation is out of order with large quantities of food rich in fat is to court disaster. In addition, mention should be made of the possible influence of delay in the institution of the hormonic regulation in the stomach of the infant on the perfect accomplishment of gastric digestion. It will thus be realized that a little is known and much more has still to be ascertained concerning the physiology of the infant loosely referred to as marasmic.

## THE HOBART HOSPITAL SCANDAL.

THE average man with solicitous feelings for the welfare of the sick poor is convinced that he is capable of managing a hospital. The fact that under ordinary circumstances the cost of maintenance is defrayed out of money contributed voluntarily or compulsorily by the income-earning members of the community seems to provide him with an excuse to seek a place on the board of management of the hospital in his district. He imagines that ordinary business instinct or ability is all that is needed in conducting the affairs of a public hospital. In consequence, our hospitals are often mismanaged and deteriorate into fifth-rate institutions.

The events leading to the McClintock inquiry should have enlightened the public of Hobart that the management of the General Hospital left much to be desired. Then followed the irresponsible action of the late Tasmanian Ministry in inducing Parliament to legislate for the still worse administration of this institution. The medical profession protested against this retrograde step and embarked

in a struggle with the Government which has unfortunately been obscured by another issue in connexion with which the Government took the unprecedented course of legalizing the registration of a practitioner by special Act of Parliament when the validity of a document purporting to be a diploma of a disreputable American college was questioned. The position in the Hospital became still more unfortunate when the Government found it expedient to pack the staff by nominees who were prepared to sacrifice the high ethical standards of the medical profession at the bidding of their paymasters. Lately two members of the medical staff, the paid Surgeon-Superintendent and the paid House Surgeon, have quarrelled. Certain charges were levelled by the former against the latter and by the latter against the former. In June of this year the grumblings became louder and the House Committee was required to intervene. The details of what appears to have been a series of unseemly outbursts of temper and of mutual incriminations have been published in the daily press of Tasmania in nauseating detail. The Board of Management received reports from the House Committee and requested the Chief Secretary to hold an inquiry by Royal Commission into the charges of insubordination alleged against the House Surgeon and into the counter charges made against the Surgeon-Superintendent. The House Committee recommended the dismissal of the House Surgeon. The Chief Secretary, no doubt with the full concurrence of his colleagues, eventually refused to adopt this course on the untenable plea that the matter was one with which the Board of Management should deal. The Government of Tasmania has been quite consistent in refusing to have the affairs connected with the Hobart General Hospital investigated to the full in an impartial and judicial manner. The Board of Management considered the reply of the Chief Secretary on September 9, 1922, and, finding no escape, shouldered the responsibility of arriving at a decision. Some of the members apparently objected to the manner in which the House Committee had presented its case and were loath to condemn the House Surgeon without a proper investigation of the facts and without affording to him an opportunity of defending himself. The majority, however, took the opposite view. They were satisfied that there had been insubordination and that the institution had suffered by the friction between the two medical officers. In the end only two members voted against a motion for the dismissal of the House Surgeon, watered down by a suggestion that he might resign if he wished. The charges made against the *protégé* of the Government seem to have disappeared.

These unsavoury happenings must damage the reputation of an institution which has already suffered during the past few years on account of the stubborn stupidity and shuffling tactics of politicians. This state of affairs is all the more intolerable because strife, bitterness and tomfoolery necessarily militate against the interests of the persons for whom the Hospital exists. The medical profession is willing and anxious to build up a useful, well-managed and up-to-date institution of which Tasmania might be proud. Its members have given their services gratuitously in the past for the benefit of the poor needing medical and surgical treatment and they could easily be induced to re-enter the Hospital in order to raise its status to a high level. The contentious question, the admission of well-to-do patients to the free wards, is not an insoluble one. It has been admitted that the needs of the necessitous are paramount. If the affluent are to be permitted to use the institution, honorary medical officers should be permitted to demand remuneration of them and to treat them in special wards or private rooms.

We are convinced that reasonable negotiations between the Government and the medical profession would lead to some form of amicable arrangement and the removal of the original cause of trouble. Sooner or later the Hospital will be rescued from its unfortunate position and come into line with the other public hospitals of the Commonwealth. The system of hospital standardization will, it is hoped, be introduced in the near future into Australia. If this were done the unsoundness of the entire system obtaining at the Hobart General Hospital would be exposed to full view. It would be wise to avoid this exposure by the adoption of a spirit of reasonableness and conciliation on the part of the Government.

## BRASS FOUNDERS' AGUE.

In all forms of metallic poisoning the physical condition of the poisonous substance and its relation to the absorbing surface are matters of the greatest importance. As a rule, toxicologists have paid close attention to the solubility of the metallic salt on the assumption that the chemical condition at the time of contact with the body is necessarily a determining factor in the production or absence of toxic effects. It has, however, been shown that the living organism is capable of dealing with any metallic salt, no matter what its chemical state, provided that it offers a sufficiently large surface for absorption. In other words, the state of division is of far greater importance than is the solubility of the compound. It has also been shown that absorption of metallic salts in a state of fine division takes place in proportion to the extent of the absorbing surface. Thus a fine, non-agglutinated powder is absorbed with great avidity from the mucous surface of the lungs, with more difficulty from the peritoneal cavity, less readily from the stomach and least easily from the subcutaneous tissue.

For many years clinicians, industrialists, toxicologists and chemists have endeavoured to arrive at a definite conclusion concerning the toxic element in the symptom complex known as brass founders' ague. The literature on this subject is large, but it is very difficult to cull from it evidence of an acceptable nature to solve this question. Mr. Philip Drinker, working in the physiological laboratories of the Harvard Medical School, has made an important contribution on zinc poisoning which should serve to clear up many of the obscure factors involved.[1] In the first place he gives a very clear account of the commercial processes in the manufacture of zinc oxide and brass. He points out that it is eminently pertinent to the problem to ascertain the physical condition of the various metals on volatilization and oxidation while in a vapourous state. The melting and boiling points of zinc, copper, lead, cadmium and other metals at varying partial pressures are discussed, as is the principle on which the commercial process of preparing zinc oxide by the volatilization of the metal. Zinc boils at between 920° C. and 930° C., while copper boils at about 2,350° C.. Brass represents an alloy of zinc and copper. When the alloy is composed of seventy-five of copper and twenty-five of zinc, the melting point is 920° C., while when the proportion is sixty of copper and forty of zinc the melting point is about 890° C.. If there is an excess of zinc, the excess is merely mixed with the alloy and does not take part in the solid solution. The melting of brass consequently occurs at the temperature of the boiling of zinc and in consequence molten brass gives off more or less volatile zinc. It has been shown by numerous observations and experiments that the symptoms of brass founders' ague appear exclusively in men who work in the immediate neighbourhood of the moulds or crucibles containing molten brass. Mr. Drinker has no doubt that this affection is a special instance of zinc poisoning. It has been suggested that the zinc is absorbed in the form of the soluble chloride and not as the inert oxide. Direct observation has revealed the fact that a certain amount of the metallic oxide is inhaled, while the greater part is caught up in the fauces and mouth and is swallowed. The workers with toxic symptoms excrete a relatively small quantity of zinc in the urine and a large amount in the fæces. All the work conducted on this subject goes to prove that swallowed zinc, except in very large quantities, is harmless. A difficulty has arisen in the acceptance of this teaching, in view of the apparent harmlessness of the handling of zinc oxide powder, which, being in a state of fine division, should be readily inhaled. Mr. Drinker offers the following explanation, which is so plausible that its acceptance should be universal. The volatilized zinc issuing from the crucible or mould oxidizes readily in the presence of a free supply of oxygen. The zinc oxide as it cools exists in the form of freshly burned particles of considerably less than 0.5 $\mu$ in diameter. Particles of this size do not obey Stokes's law governing the rate of settling and in consequence they remain dry, discrete and suspended in air. If inhaled at this stage they follow the current of air and do not tend to agglutinate. On the other hand, zinc oxide powder that has been collected, has undergone a change. Moisture has been adsorbed on to the surface. The particles reach 0.5 $\mu$ in size and flocculation occurs, which means that the particles adhere to each other. The condition of the ordinary zinc oxide powder is therefore far less favourable for absorption than that of the freshly burned oxide. Owing to the dispersion and the absence of adsorbed moisture the latter offers an immense surface for the respiratory mucous membrane to attack. Under such favourable circumstances the particles will not remain deposited on the mucous surface for long, but will pass into the tissues either in particulate form or in solution. Since the albuminate of zinc is insoluble, it is not improbable that the chloride is formed in the tissues. Mr. Drinker favours this view, but apparently neither he nor anyone else has ascertained whether the zinc on absorption exists in organic combination or as free kations.

In the course of his review of the literature he touches lightly on a very important point to which but scant attention is paid. It is estimated that workers suffering from brass founders' ague are seized with symptoms after breathing air containing 0.23 milligramme of zinc oxide per litre of air for one hour. On the computation that a man breathes about half a litre at each respiration seventeen times a minute, the amount of zinc inhaled would be about 120 milligrammes. Mr. Drinker calls attention to the fact that a man undergoing vigorous exercise breathes twice or three times this amount of air. It may therefore be estimated that the worker would receive between 250 and 350 milligrammes of zinc oxide, of which perhaps one-quarter would reach the lungs and be absorbed. Relatively small amounts are recovered in the urine. It would thus appear that the symptom-producing dose would be under two milligrammes per kilogram of body weight. If these figures correspond to the actual state of affairs, it is surprising that zinc salts under ordinary circumstances exert so little toxic effect.

[1] *The Journal of Industrial Hygiene*, August, 1922.

## THE NATURE OF MYASTHENIA GRAVIS.

Although many observers in the latter half of the nineteenth century, including Wilks, Goldflam, Erb, Hoppe and Jolly, described certain disturbances in neuro-muscular function now known as *myasthenia gravis*, it was not until Strumpel in 1895 and Campbell and Bramwell in 1900 reviewed the literature and recorded their observations that the condition became generally known and was regarded as a definite pathological entity. The disease is a rare one, but Bramwell has pointed out that it probably occurs oftener than is supposed. He states that the condition, as manifested in some patients, is frequently diagnosed as hysteria and in this way is often missed. The causation has not been definitely determined. The early observers regarded it as a bulbar paralysis and called it asthenic bulbar palsy. No definite lesions, however, were discovered in the bulb with the exception of minute hæmorrhages. The hæmorrhages were recent and the fact that death in *myasthenia gravis* is generally due to asphyxia probably accounted for their appearance.

Oppenheim endeavoured to explain it as a congenital condition and many observers drew attention to the fact that it frequently appears after infective diseases. The occurrence of tumours of the thymus gland with metastatic deposits has been held responsible for its appearance and it has frequently been noticed as an accompaniment to Graves's disease. None of these hypotheses, however, has offered any satisfactory explanation of the ætiology of the disease. In regard to the histological changes, those most constantly present are in the muscles in the form of deposits of mononuclear lymphocyte-like cells. Farquhar Buzzard has styled these deposits "lymphorrhages." Their most constant appearance is in the muscular tissue, but careful examination of the structure of other organs will frequently reveal their presence. The muscle fibres themselves commonly display early changes in the form of plasmatic swelling, proliferation of the nuclei of the sarcolemma and occasionally hyaline and granular degeneration. The electrical response of the muscles is altered. The muscular contractions diminish rapidly in response to faradism, while they continue to act normally to galvanism. From this Bramwell concludes that it is the nerve fibres and not the muscle fibres that are actually functionless. He thought that this state of the motor neurones might be due to a congenital defect in the neurones themselves or that it was the result of the action of a toxin caused by some metabolic defect. In this connexion it is interesting to note that female patients suffering from *myasthenia gravis* apparently recover during pregnancy. One of Goldflam's patients stated that to be always well, she would have to be always pregnant. It has been supposed that the *fœtus in utero* is able to supply something lacking in the metabolism of the mother. Kaufmann in 1906 held that *myasthenia gravis* could be ascribed to a metabolic insufficiency. Several observers have reported a retention of nitrogen and loss of calcium with a low creatin excretion while the patients were on a creatin-creatinin-free diet.

In view of these observations, Dr. B. W. Williams and Dr. S. C. Dyke have recently conducted an investigation in order to ascertain whether or not disturbance of creatin metabolism was a common occurrence in *myasthenia gravis*.[1] They investigated the metabolism of four patients afflicted with the disease. A creatin-creatinin-free diet, as recommended by Sprigg, was adopted and the patients were kept on the diet three days before observations were begun. Normally creatin does not appear in the urine. Creatin given by mouth to a healthy individual is excreted as creatinin. In all four patients on a creatin-creatinin-free diet creatinuria was present. The amounts of creatin excreted in grammes *per diem* by the patients averaged respectively 0.047, 0.051, 0.047 and 0.449. Small pieces of muscle were excised from two of the patients for the purposes of creatin estimation. About six grammes were removed from the *tensor fasciæ femoris* muscle in each instance. The normal percentage of creatin in human muscle is 0.3. In the two samples of muscle examined the percentages were 0.21 and 0.25.

Drs. Williams and Dyke also made some observations on the carbo-hydrate metabolism. They point out that Maclean has shown that during the stage of lowering of the blood-sugar concentration in a normal individual after a dose of glucose the arterial and capillary blood contains more sugar than the venous blood. They found that in *myasthenia gravis* this difference does not exist. Sugar tolerance tests were carried out on three of the patients. They found that doses of fifty grammes of glucose produced little change from the normal blood-sugar curve. After a dose of one hundred grammes there was a great difference in the curves from those of a normal individual. The curves were much higher and a definite glycosuria was present in all. After the ingestion of levulose no incerase in blood-sugar occurred and hence they regarded the function of the liver in regard to blood-sugar as normal. They conclude that the defect in the carbo-hydrate metabolism is probably located in the muscles.

These observations, although unfortunately limited to a few cases, are a valuable addition to a subject of great difficulty. They certainly tend to show that the solution lies in the path of metabolic activity and departure from its normal state and indicate the lines along which future work will be undertaken.

## AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

The Executive Committee of the Australasian Medical Congress (British Medical Association) has drawn up a general programme of office-bearers, committees and sections for the First Session to be held in Melbourne in November, 1923. This "Organization" will be published in full in The Medical Journal of Australia of October 14, 1922. The Executive Committee invite members of the six Australian Branches and of the New Zealand Branch of the British Medical Association to attend this Session and to contribute scientific papers, in order that its success may be achieved. The leading note of the Congress will be preventive medicine.

[1] *The Quarterly Journal of Medicine*, July, 1922.

# Abstracts from Current Medical Literature.

## THERAPEUTICS.

### The Elimination of Strychnine in the Urine.

SOMA WEISS AND ROBERT A. HATCHER (*The Journal of Pharmacology and Experimental Therapeutics*, July, 1922) have recorded *inter alia* certain observations on the elimination of strychnine in the urine of man. Strychnine sulphate was administered orally and intramuscularly in single doses of four milligrammes and in repeated oral doses amounting to about fifteen milligrammes over a period of twenty-four hours. The urine was collected at periods of six hours, twelve hours and twenty-four hours and the amounts of strychnine present were estimated. It was found that the kidneys excrete amounts equal to 20% of that administered in a single dose and a much lower percentage of larger doses taken through the mouth over periods of twelve and twenty-four hours respectively The percentage of the strychnine eliminated by the kidneys is a measure of the eliminative efficiency of the liver rather than that of the kidneys, since the latter excrete only that complement of strychnine which the liver fails to excrete. Diuresis hastens the elimination of strychnine by the kidneys, but it does not necessarily increase the total amount eliminated in the urine after the intramuscular injection of a single dose. In fact, it may be attended with the renal elimination of a smaller total than would occur in a similar experiment without diuresis. In cases of acute poisoning with strychnine it would appear that the liver was the principal protective organ, the kidney being concerned mainly with the excretion of traces of the poison which re-enter the circulation after temporary fixation in tissues incapable of destroying the poison. The authors point out that their observations are of interest in view of the opinion formerly held that strychnine was eliminated almost entirely by the kidneys. They have been unable to find any experimental evidence to support that view and quote against it the findings of Hatcher and Eggleston, who were able to recover in the urine and fæces only a small fraction of the strychnine administered to cats, dogs and guinea-pigs, the larger part being destroyed in the body. Hatcher and his former colleague also found that if strychnine be added to diluted defibrinated blood and perfused through the liver, the poison is destroyed by that organ.

### Oxygen Therapy.

A. L. BARACH AND M. N. WOODWELL (*Archives of Internal Medicine*, October, 1921) have reviewed the history and published the results of their experiments on the effects of the administration of oxygen in heart failure, lobar pneumonia and *encephalitis lethargica*. One of the first symptoms of oxygen want in normal subjects is periodic breathing. Impairment of mental functions, nausea and headache appear later and often vomiting and diarrhœa. Cyanosis is *prima facie* evidence of lack of oxygen in clinical disease. Oxygen has been administered by means of masks, nasal catheters, oxygen chambers and oxygen bed tents. In these experiments a face mask was used connected through a soda-lime canister with a re-breathing bag in turn connected with an oxygen tank. In those patients who could not stand the mask, a soft, flanged rubber rubber tube replaced the mask, the tube being held in the mouth. The oxygen saturation of the blood was determined by Van Slyke's method before and after each experiment. It was found that inhalation of oxygen for one-half to two hours by seven patients with cardiac insufficiency increased the arterial saturation, relieved cyanosis and slowed the pulse. The patients said they felt more comfortable, but they were rarely enthusiastic. In eleven patients with lobar pneumonia and four with broncho-pneumonia cyanosis was relieved, the pulse was slowed, the mental condition was frequently improved, but the dyspnœa was usually not relieved. The effect of a single administration was usually temporary; the effect of repeated and prolonged administration was a general beneficial change. The prolonged administration of oxygen to three patients with acute oxygen want associated with pulmonary œdema resulted in a striking clinical improvement and seemed to avert a fatal issue. In two patients with *lethargic encephalitis*, who were comatose, cyanosed and had extremely shallow breathing, oxygen inhalation relieved the anoxæmia, but was without effect on the steady accumulation of carbon dioxide, which apparently caused progressive cardiac failure, shallow respiration, interfering with oxygen absorption and carbon dioxide elimination.

### Quinidine.

W. W. HAMBERGER AND W. S. PRIEST, JUN. (*The Journal of the American Medical Association*, July 15, 1922) record their results to date of the treatment of eighteen patients suffering from auricular fibrillation with quinidine. Eleven of these patients had the normal heart rhythm restored and in five of these it lasted three months or more. In seven patients regular rhythm was not established by quinidine treatment; in some patients in whom regular rhythm occurred after quinidine administration, auricular fibrillation recurred one or more times. The authors state that the patients who respond best, are those in whom fibrillation is of recent onset and who have no signs of decompensation. The most unfavourable subjects are those with long-standing fibrillation and advanced heart failuree. The dose used was 0.2 to 0.6 gramme three or four times a day or 0.2 gramme every two hours for four days, increased to 0.4 gramme every two hours if there had been no good effect. After the establishment of sinus rhythm 0.2 gramme once or twice a day was used for a considerable period. Quinidine may usually be given without a preliminary course of digitalis.

### Pneumococcus Antibody.

R. L. CECIL AND N. P. LARSEN (*The Journal of the American Medical Association*, July 29, 1922) reviewed one thousand cases of lobar pneumonia and have reported the results of treatment with pneumococcus antibody solution. Some type of pneumococcus was present in the sputum of 90% of the patients. Four hundred and twenty-four patients were treated with pneumococcus antibody solution given intravenously (fifty to one hundred cubic centimetres once, twice or three times a day) until the temperature remained below 37.8° C. (100° F.); an average of two hundred and twenty-five cubic centimetres was administered per patient. Four hundred and ten patients with lobar pneumonia were treated without antibody solution. It was found that the death rate in those given the specific treatment was 21.4%; in the controls it was 28.3%. The best effect was produced in pneumococcus type I. pneumonia. The solution used was a serum-free solution of pneumococcus antibody.

### Pyelitis.

H. F. HELMHOLTZ (*The Journal of the American Medical Association*, July 22, 1922) has studied the action of alkalies, hexamethylenamin and phenyl salicylate (salol) in pyelitis. Treatment with alkalies does not produce a higher degree of alkalinity than pH 8.6 in the urine of human beings, whereas the growth of colon bacilli is not inhibited until the H-ion concentration reaches 8.2 to 9.6. The author, therefore, considers that alkalies have no direct specific action on colon bacilli in the urine; any good effects produced by them must be due to some other cause, possibly diuresis. Hexamethylenamin has a specific effect on colon bacilli in the bladder when the urine is acid, but there is no proof that it affects urine in the renal pelvis in infections due to the *Bacillus coli communis*. Salol appears to have no beneficial effect on pyelitis.

### Quinine.

K. MCLAY (*The Journal of the Royal Army Medical Corps*, February, 1922), in a study of malaria in Macedonia, reports the effects produced by administration of quinine orally, intramuscularly and subcutaneously. He concludes that the best results are attained by oral administration. The other two methods investigated caused much local reaction, pain and even abscess formation and presented no increased therapeutic action in comparison with the oral method. Observations on the benign tertian parasite indicate that quinine has an immediate effect on the malarial parasite, not only on the young sporulating merozoite, but also on the intracorpuscular stage.

## UROLOGY.

### Ureteric Reflux in Renal Tuberculosis.

ANDRÉ AND GRANDINEAU (*Journal d'Urologie Medicale et Chirurgicale*, July, 1921) publish the histories of five patients in whom, with a unilateral renal tuberculosis, reflux of urine from the bladder up to the healthy kidney was observed. It has been established elsewhere that the conditions favouring such reflux, whether in health or disease, are a certain amount of tonicity of the bladder wall and a sudden increase in the intravesical pressure. It will readily be understood that these factors are constantly present in the irritable tuberculous bladder. In addition, insufficiency of ureteric mouth closure on the healthy side is apt to result from infiltration of the bladder muscle surrounding the orifice or of the muscle of the trigone. Under such conditions these muscles contract poorly and exert very little sphincteric action on the ureteric mouth. This may be seen at times as a patulous orifice, in spite of the fact that no ulcers or tubercles immediately surround it. The refluxes were observed by means of ureterograms and also by the occurrence of pain in the healthy kidney during urination or painful contractions of the bladder. In one instance the ureter on the "healthy" side was seen at autopsy to be dilated and tortuous, the pelvis and calyces were dilated, the kidney and upper ureter were entirely free from tuberculosis, while the ureter in its lower five centimetres was definitely tuberculous. Reflux on the diseased side is uncommon on account of the frequent tuberculous stricture occurring on that side. On account of the gaping of the meatus on the healthy side in cases of reflux and the occasional collection of regurgitated pus and tubercle bacilli from the sound kidney, the diagnosis of bilateral renal tuberculosis may be made when in reality one kidney alone is dilated. The greatest difficulty arises when the efficiency of the latter organ is found to be low on account of the dilatation. When it is certain that the lowered efficiency on the better side is merely the result of the ureteric reflux, it is advisable to secure amelioration by a small tube nephrostomy before proceeding to nephrectomy of the tuberculous kidney.

### Genito-Urinary Reflexes.

J. M. BARTRINA (*Journal d'Urologie Medicale et Chirurgicale*, May, 1922) studies the reflexes manifested in the genital and digestive tracts during the course of urinary diseases, as well as reflexes in the badder arising from renal and ureteral lesions. The outstanding example of a reno-vesical reflex is the vesical irritability (cystalgia) which not seldom precedes all other discoverable signs in renal tuberculosis. Uretero-prostatic and ureteropenile reflexes are seen occasionally in calculous disease of the kidney and ureter. The outstanding feature may be pain in the perineum or along the urethra and this may mislead the clinician. The observer may also be led astray by noting a urethral discharge which is due to reflex modification of innervation and circulation. Bartrina considers that a reflex from a seemingly insignificant and negligible lesion of the kidney may explain the vesical and urethral distress observed in some cases of gout, diabetes, oxaluria and phosphaturia; he does not think that the alteration in the urine will account entirely for the symptoms in this group He adduces Albarran's observations in instances of unilateral renal calculus, where the opposite kidney, though sound to the eye, was already damaged by some factor dependent on the uratic, oxalic or other diathesis. In the alimentary tract reflexes arising from the urinary tract are seen and thus in kidney stone and renal ptosis the predominating symptoms may be constipation, spasms of intestinal colic or even the symptoms of partial obstruction. The linking up of these diverse regions by nerves is by means of the wide connexions of the inferior mesenteric sympathetic ganglion, ascending fibres to the renal plexus, lateral fibres to the sympathetic chain, arterial fibres along the whole distribution of the inferior mesenteric artery and descending branches to join the pelvic plexus and so supply the bladder, genital organs and urethra.

### Radium in Prostatic Cancer.

H. G. BUGBEE (*Journal of Urology*, December, 1921) records his experiences with the more recent methods of applying radium in cancer of the prostate. Most of the patients were observed during fifteen months after treatment and the results, with better methods of treatment, have been very different from those in his earlier series of cases, dating back now to fifteen years. The histories of seventeen patients are detailed. The methods from which the author has seen most benefit derived are, firstly, radium needles introduced into the prostate by suprapubic approach; secondly, needles passed into the gland from below after perineal dissection; thirdly, surface applications, either through the urethra or the rectum, made later on to supplement the action of the first or second method.

### Pyelonephritis.

O. C. FOOTE (*Californian State Journal of Medicine*, April, 1922) publishes a critical review of one hundred cases of pyelonephritis. The series excludes pyelonephritis in children and infections secondary to urinary calculus or obstruction in the lower urinary tract. This narrows the ætiology to infections secondary to ureteric obstructions, to malpositions of the kidney, to displacements of the female pelvic organs and to gastro-intestinal disturbances. Sixty-nine of the patients were females. In sixty both kidneys were affected; in eighteen the right organ and in twenty-two the left alone was diseased. An infection due to a bacillus was present in eighty-four, while a coccus was the causal organism in sixteen. The large majority of patients complained of bladder disturbance only; a few suffered from renal aching. The pain may be low down and on the right side may simulate that of appendicitis. The latter disease may co-exist. Pus was present in the urine of ninety-five patients and the other five displayed pure bacteriuria. Stained smears made at once from ureteric catheter specimens were considered of more accurate diagnostic value than cultures. Blood was present in the urine of fifty-nine patients and sixty-five had albuminuria. Treatment was directed towards the ætiological factors enumerated and, in addition, local treatment by pelvic lavage was found of value.

### A New Method of Perineal Prostatectomy.

J. T. GERAGHTY (*The Journal of Urology*, May, 1922) publishes a preliminary report on a modification of the Young method of perineal prostatectomy. He states that in the usual perineal operation the sphincter of the membranous urethra is either dislocated or divided before the membranous urethra can be opened. Faulty urinary control, especially after operating on large prostates, is not always eliminated even by experts in the perineal method. The author's method is to avoid injuring the external sphincter by using a long prostatic tractor designed by Dr. Henry Freiberg. This tractor is long and curved and is designed to be passed into the bladder through the whole length of the urethra. The remaining steps of the operation are similar to Young's method, except that the incision in Devonvillier's fascia is of a blunt inverted V shape with the point of the V near the apex of the prostate. The whole gland is shelled out if possible in one mass.

### Lympho-Cystic Urethral Lesions.

P. S. PELOUZE (*The Journal of Urology*, March, 1922) presents his conclusions on posterior urethral and bladder-neck cysts causing burning on urination. His contention is that patients showing these lesions have somewhere in the body an active focus of tuberculosis. The cysts are small, never ulcerate or rupture spontaneously and are usually situated posterior to the *veru montanum* on the floor of the lateral walls of the urethra; sometimes they are at the vesical neck. The lesion arises as a small, sub-mucous accumulation of lymphoid cells. Liquefaction ensues and, if a cyst be ruptured by instrumentation, a milky fluid is seen to escape. The local discomfort caused by the cysts is mild. It is usual to find an active focus of tuberculosis in some other part of the body, generally the lungs. The cysts either recur or are uninfluenced by local treatment so long as the tuberculous focus is untreated. There is, however, good reason to believe that the cysts themselves are only exceptionally of tuberculous structure.

# British Medical Association News.

## SCIENTIFIC.

A MEETING of the Western Australian Branch of the British Medical Association was held at the Perth Hospital on August 16, 1922, DR. D. M. MCWHAE, C.M.G., C.B.E., in the absence of the President, in the chair.

### Splenic Anæmia.

DR. D. M. MCWHAE presented a child, aged three years. The child had been pale since birth. Dr. McWhae had seen the child for the first time in December, 1921, when the spleen had been just palpable. At the time of the demonstration the lower border of the spleen was five centimetres below the costal margin. The liver was beginning to enlarge. The blood examination had revealed the following resultts:

| | |
|---|---|
| Red blood corpuscles .. .. | 1,170,000 per cubic mm. |
| White blood corpuscles .. | 5,900 per cubic mm. |
| Poly-morpho-nuclear cells.. .. .. | 36% |
| Lymphocytes .. .. .. .. .. .. | 58% |
| Large mono-nuclear cells .. .. .. | 6% |
| Eosinophile cells .. .. .. .. .. | 0 |

There were a few nucleated red corpuscles. Dr. McWhae had therefore decided that leuchæmia was not present. No hæmorrhages had occurred to account for the anæmia. He found that the disability was Banti's disease and the commencing enlargement of the liver indicated that the second stage was beginning. He stated that, if untreated, the patient would eventually enter the third stage of the disease, with cirrhosis of the liver, ascites and death. He proposed to perform splenectomy for the purpose of retarding or even curing the disease. The operation should be undertaken before serious structural changes occurred in the liver. He referred to another patient whom he had under observation. The spleen had been removed on account of splenic anæmia three years previously, notwithstanding the presence of great enlargement of the liver and ascites. The patient had probably had the Gaucher type of the disease. He was in moderately good health and was attending school, but his liver was much enlarged.

### Spleno-Medullary Leuchæmia.

DR. MCWHAE'S second patient was a man, aged twenty-five years, who had been seen for the first time on June 16, 1922. Nine months before that date he had had an attack of abdominal pain and vomiting. Since then the pain had recurred at frequent intervals. The man was distinctly anæmic and the spleen was greatly enlarged, reaching below the brim of the pelvis and extending five centimetres beyond the middle line. The examination of the blood had yielded the following:

| | |
|---|---|
| Red blood corpuscles .. .. | 4,200,000 per cubic mm. |
| White blood corpuscles .. | 325,000 per cubic mm. |
| Colour index.. .. .. .. .. .. .. | 0.95 |

Of the white cells, 60% were myelocytes. He stated that in spleno-medullary leuchæmia splenectomy was always followed by disastrous results; it should not be undertaken. He held that in blood diseases which appeared to be splenic anæmia, repeated examination of the blood should be carried out in order to exclude atypical forms of leuchæmia before splenectomy was attempted. The patient exhibited had been treated by means of X-rays with weekly exposures of the spleen and the long bones. He had also been given arsenic internally. The spleen had become considerably reduced in size and the number of white corpuscles had decreased to 25,000 per cubic millimetre. He had formed the opinion that the treatment was producing a remission. He admitted that X-ray treatment was merely palliative, but he claimed that it prolonged life. Usually the patients died in from three to five years, but he had had one patient who had spleno-medullary leuchæmia for eight years. It had been reported that a patient had remained alive and relatively well for thirteen years. Benzol sometimes produced striking results, reducing the size of the spleen and diminishing the number of white blood corpuscles. It was not free from danger. One of his patients had had a cerebral hæmorrhage while undergoing treatment with this drug.

### Bulbar Paralysis.

DR. MCWHAE presented a man, aged forty-eight years, whom he had seen for the first time two months previously. After an operation eighteen months before he had lost his speech suddenly for a period of seven days. Nine months before admission his speech had become husky and indistinct. It had gradually become worse. The patient could not whistle, owing to weakness of the lips; his face was expressionless as a result of paralysis of the facial nerve. The tongue was wasted and furrowed and showed fibrillary twitching. Fluid when swallowed occasionally regurgitated through the nose. The condition was bulbar paralysis. There was also wasting of the interossei muscles of the hands. The legs and arms were slightly stiff. All the deep reflexes were much exaggerated and both plantar reflexes were extensor in their response. The degeneration of the motor nuclei had therefore not only involved the bulbar nuclei, but had spread to the nuclei of the nerve fibres of the pyramidal tracts and to the motor nuclei in the cervical portion of the spinal cord. This constituted a form of progressive muscular atrophy called amyotrophic lateral sclerosis.

The next patient was a married woman, aged forty-eight years, who had been admitted in the middle of July, 1922. She was suffering from bulbar paralysis, but the degeneration had not spread to any great extent from the bulbar region. She was very emotional and childish and was approaching a final condition of dementia. It was interesting to note that it had been thought that both these patients in the early stages of their disease had been suffering from functional trouble.

### Bronzed Diabetes.

DR. MCWHAE showed a man, aged forty-seven years, who had been admitted to hospital two months previously. His skin was deeply pigmented. His liver was enlarged. He was passing sugar in his urine, save when he was dieted in regard to fats and carbo-hydrates. There was no asthenia. His blood pressure was raised (systolic 170 millimetres and diastolic 110 millimetres). There was no pigmentation of the mucous membranes. Dr. McWhae concluded that he was not suffering from Addison's disease. He believed that the condition was due to hæmo-chromatosis in which an iron-containing pigment was deposited in the liver, pancreas, spleen, skin and elsewhere. Cirrhosis of the liver and pancreas were produced by this condition and the fibrotic change in the pancreas involved the islands of Langerhans and produced diabetes.

### Diabetes Mellitus.

DR. MCWHAE then presented a young diabetic, aged sixteen years, who had lost nineteen kilograms (three stone) in weight during the past six months. She had been admitted to hospital on July 11, 1922, complaining of pains and weakness in the legs. Under dietetic treatment she had done well in the short time. Her urine had been rendered free of sugar with a diet containing twenty-two grammes of carbo-hydrate, eighty-seven grammes of protein and forty-two grammes of fat, *i.e.*, an energy value of 18.7 calories per kilogram of body weight. The diet would be steadily increased. Attention was directed to the patient on account of the grave prognosis of diabetes in persons of her age. He proposed to report the ultimate result of treatment at a later date.

### Heart Block.

The next patient was a man, aged fifty years. He had been rejected when he had offered his services with the Australian Imperial Force in 1914 on account of weakness of the heart. He had remained quite well until six weeks before admission to hospital on June 24, 1922. He had then been giddy and weak. The pulse was twenty-eight per minute. The polygraphic tracings showed that there were two auricular contractions to one ventricular contraction.[1]

[1] Two hours later the patient died suddenly. It was found at autopsy that his heart was large and there were signs of heart failure, *viz.*, some fluid in the abdominal cavity and passive congestion of the liver and spleen. The aortic valves were much thickened and calcified from old endocarditis. In the auriculo-ventricular region about the bundle of His the tissues had undergone calcification, probably from old mural endocarditis. The passage along the bundle of His of the wave to the ventricle from the auricular systole had been completely blocked by the degenerated tissue.

### Radiographic Exhibits.

DR. DONALD I. R. SMITH exhibited numerous skiagrams. The series included a dental film revealing the congenital absence of the right upper lateral incisor in a boy aged twelve years. The second right upper bicuspid had been extracted. The second skiagram depicted the septic relic of the lower jaw of twenty years' standing. The third was a shadow picture of a calculus impacted in the right ureter. The patient was a man, aged fifty years, stout and sallow. He had complained of pain in the right side of the abdomen for six weeks, associated with vomiting. This had been followed by absence of symptoms for a period of six weeks. At the end of this time the pain had recurred for seven days. It had radiated to the groin and penis, but not to the right scapular region. There had been hæmaturia. In one film the kidneys and ureters had appeared natural, but in another an ovoid calculus impacted in the lower end of the ureter was plainly visible. The patient had passed a number of gall stones. While in hospital he had had rigors and fever and tenderness had been detected over the gall bladder. Dr. Smith attributed the attacks of pain to the ureteric calculus.

After he had shown a picture of cervical ribs, he displayed a skiagram illustrating a mid-carpal fracture-dislocation. He stated that this was a rare injury. He had seen six instances in eight years. At times it accompanied fracture of the scaphoid bone and was produced by the continuance of the violence after the scaphoid had broken. The four bones of the distal row, together with the scaphoid, were carried backward, while the inner half of the scaphoid, the semilunar, the cuneiform and the pisiform bones remained in position. If untreated, this injury resulted in complete loss of the function of the wrist.

He also showed a skiagram of an arm that had suffered another rare injury. The ulna had been fractured and the radius had been dislocated, but not fractured. The orbicularis ligaments had become involved in a *fibrositis ossificans*. Three films were shown illustrating the condition known as *osteitis fibro-cystica*. The condition was a mild, chronic, septic invasion of the bone following a bruise without fracture. It resulted in rarefying cyst-like cavities appearing in the medulla or in the cancelous tissues with condensed laminæ surrounding these cavities representing an attempt at compensation. The bone subsequently became enlarged and showed cortical thickening, which he interpreted as a further endeavour to maintain its function. When the disease was well established, very slight violence would suffice to produce a "spontaneous fracture." Dr. Smith regarded the condition as related to Paget's disease.

He also exhibited skiagrams of a psoas abscess extending to the thoracic spinal region of a child, a congenital dislocation of the hip, osteitis of the head of the humerus, with areas of rarefaction and surrounding condensation in the centre of the bone, Pott's disease in a young child, a small sequestrum in the left ala of the sacrum, with reaction of bone indicative of a mild, chronic septic process, a local patch of rarefying and necrotic osteitis without reaction, probably tuberculous in nature, an osteo-chondroma of the lower end of the ulna, myeloid sarcoma of the jaw, osteoma of the *os calcis* causing a painful heel, early tuberculous disease of the hip, advanced tuberculous disease of the hip, septic arthritis producing destruction of the shoulder joint, caused by gross oral sepsis, septic arthritis of the toes caused by suppuration in the apices of teeth, destructive arthritis of the inferior radio-ulnar joint and necrosis of the radius caused by chronic gonorrhœal infection of the *cervix uteri* and typical osteo-arthritis of long duration.

Finally Dr. Smith demonstrated a skiagram of the abdomen of a man, aged twenty-nine years, who had had pneumonia six weeks prior to admission. A short time later he had noticed cramp-like sensations in his right leg and prominent veins on the inner side of the right thigh. The whole of the right thigh and groin had subsequently become swollen and painful. Five days later the swelling had appeared in the left leg. He had complained of a dragging pain in the lower region of the abdomen, which did not seem to be related to the ingestion of food. On examination some tenderness had been detected in the lower part of the abdomen and an elongated, hard swelling had been felt to the right of the umbilicus. The superficial veins of the abdomen and thorax were dilated. Five days after admission the patient had pneumonia involving the lower lobe of the right lung. After recovery from the pneumonia, the œdema of the legs had subsided. The skiagram revealed vague shadows of tumour-like outline on the right side of the body of the fourth lumbar vertebra. The area was about the size of a hen's egg. No abnormal signs were detected in the spine. The diagnosis appeared to lie between a chondroma and a calcarious deposit in a blood clot.

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A MEETING of the Section of Pædiatrics of the New South Wales Branch of the British Medical Association was held at the B.M.A. Building, 30 to 34, Elizabeth Street, Sydney, on August 18, 1922, DR. C. P. B. CLUBBE, the President, in the chair.

### Nutritional Disorders of Infancy.

DR. J. MACDONALD GILL read a paper on the nutritional disorders of infancy.

DR. MARGARET P. HARPER read a paper on "Breast Feeding of Infants" (see page 373).

DR. L. R. PARKER read a paper entitled "Nutritional Diseases of Infants and Their Treatment" (see page 377).

DR. C. P. B. CLUBBE thanked the speakers and congratulated them on the interesting and valuable matter presented.

PROFESSOR A. E. MILLS remarked on the eloquence displayed by Dr. Parker in particular. He was, however, opposed to the glandular hypothesis of the ætiology of marasmic conditions. No evidence had been adduced and if these views were accepted, they would lead to an erroneous basis for treatment. From his long association with the Ashfield Infants' Home he felt no fear of failure in rearing a baby from birth quite satisfactorily on artificial foods. Their aims and objects should be to prevent by right feeding methods digestive disorders rather than to cure them. This was the only way to rear robust children. He expressed pleasure in learning of the improvement in the infantile mortality as recorded by Dr. Gill, but still the figures were tragic. He did not pay much attention to Dr. Harper's belief in the value of checking the food requirements by calculating calories. Adult stomachs varied considerably in the time of digestion and of emptying; in children there was also a great variation in the capacity for food. Some individuals were hungrier than others. It was cruel and unnecessary to keep children waiting for the clock to indicate the time for feeding. Children were in no way different from young puppies or kittens and should, like them, be fed when they showed signs of hunger. Babies were simply young animals and should be fed like them, when they wanted it. "Fill them up, fill them up and keep them full!" should be their motto. Professor Mills approved of Dr. Parker's method of making whey, as it included a higher percentage of fat than ordinary whey. Infant feeding was a process of education and digestion could be trained as muscles were trained. A year-old baby should have plenty of meat, eggs, custard and other solid food. He denied that in the University of Sydney the teachers had failed to instruct students properly in the care of infants and children. He hoped that the Royal Alexandra Hospital for Children would soon become a clinical school in connexion with the University.

DR. HARVEY SUTTON, O.B.E., called attention to an important phase of infant mortality. During the first month, unfortunately, the death rate showed little or no improvement. The neo-natal period was beginning to receive the attention it warranted, but much more required to be done. Pre-maternity work should be developed at all the maternity hospitals. In his opinion, syphilis must play an important part in the causation of marasmic states and congenital debility, as well as of still births and early deaths. Such improvement as was manifest in the infantile mortality rates was largely due to the wide-spread and general education of women. He found it of little value to train young girls of school age in mothercraft, but he thought that much might be accomplished by organizing teaching of young women in early adolescence.

DR. R. A. GREEN advocated regular feeding, every three or four hours. His three years' experience at the Newtown clinic had established the fact that too frequent or irregu-

lar feeding was one of the most important causes of difficulty and failure in breast feeding. By establishing habits of regularity with ample time for digestion, many mothers were encouraged to continue breast feeding successfully. Under the present social and industrial conditions of a large city the feeding of children by the cry instead of by the clock courted disaster. Moreover, regularity permitted the busy mother more freedom and leisure, an important factor in popularizing maternal nursing. Medical students were not properly instructed in the art and science of breast feeding, the result being that when, as young practitioners, they were faced with some difficulty of lactation, they were handicapped and at a loss.

DR. KATE KNOWLES was delighted to learn that the Royal Alexandra Hospital for Children was to be linked up with the University for teaching purposes, because she had been favourably impressed by a similar system of training students at Great Ormond Street. Dr. Still, one of the authorities on children's diseases, had always emphasized the necessity for differentiating between the various cries of babyhood—hunger, pain, fretfulness, etc.. Dr. Knowles was whole-heartedly in favour of regularity of nursing, as crying provoked by indigestion was particularly common.

DR. H. LEAVER was eager to know if the administration of placental extracts in the early days of lactation had proved of benefit as a galactogogue. Many animals and some savages make a practice of eating the placenta.

DR. E. SELWYN HARRISON pointed out that the most important cause of nutritional disorders of infancy, excluding the true infective diarrhœas, colitis and dysentery, was wrong feeding with resulting food injuries. In breast-fed babies the source of trouble was almost always too frequent and irregular feeding. If this were corrected, the child recovered in nearly every instance. In feeding from the breast or the bottle, the milk trickled into the stomach drop by drop and was instantly transformed into curds and whey. Meara had demonstrated some years before that a runnel was formed by muscular contraction, permitting the whey to flow into the duodenum *pari passu* with the entry of milk into the stomach. A mass of curd was left, corresponding approximately to one-tenth of the volume of the total feed. It had been shown that this curd required two hours for complete digestion. A period of rest was required for proper functioning. If the child were fed more often, or excessive heat or some pareeteral infection slowed the process and another feed were poured on to the non-digested nucleus and then another and so on, the nucleus would remain untouched, as digestion attacked from the periphery of the mass of curds inwards. Fermentation, putrefaction and bacterial overgrowth occurred with production of irritating acids and toxins. In the milder stages, so often seen in breast-fed infants, little happened except the irritation of the sensory nerves of the gastric and intestinal mucosa, accompanied by pain and colic. The resulting cry was, of course, attributed to hunger and the unfortunate baby was fed before time. The warm, bland milk temporarily relieved the pain and the crying ceased. The mother therefore was convinced that the baby was hungry. The vicious circle started in this way. More colic and more crying demanded more frequent feeds, which led to more and more colic, until the distracted mother declared that her milk was "windy" and "not satisfying" and that "the child is always tugging at her." If the process continued a stage further, Nature attempted to relieve the situation by vomiting. The irritating acids and toxins also overactivitated the motor and secretory nerves with resulting increased peristalsis and out-pouring of fluid. Hence the more frequent green or watery motions. Elimination and dilution were acomplished thereby. He was therefore driven to the conclusion that the one great cause of unnecessary weaning was too frequent or irregular feeding. Regular intervals of three or four hours would prevent or correct the trouble in a very high percentage of cases.

DR. MACDONALD GILL replied briefly. He stated that, as far as he was aware, there were no reliable statistics concerning syphilis as an ætiological factor in gastro-enteritis. The Wassermann test carried out in the Children's Hospital had been disappointing.

DR. PARKER stated that he had not suggested definite glandular deficiency, but rather delayed glandular development, particularly in the digestive tract, as underlying marasmus and allied conditions.

## CORRIGENDUM.

IN the amended Articles and By-Laws of the British Medical Association printed on pages 364 to 367 of our issue of last week the final word "Council" in Article 7, "Re-eligibility," should have been cancelled.

## NOTICES.

THE COUNCIL OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION has arranged the following provisional programme of the Branch meetings. The Scientific Committee reserves to itself the right to modify the arrangements, but it is hoped that no changes will be necessary.

**October 4, 1922.**

*At the Walter and Eliza Hall Institute of Research in Pathology and Medicine, Melbourne Hospital, at 8.15 p.m.*

DR. H. DOUGLAS STEPHENS: "Clinical Symptoms of Enlargement of the Spleen in Children."

DR. S. O. COWEN: "Familial Splenomegaly."

DR. K. HILLER will open the discussion.

**November 1, 1922.**

CLINICAL MEETING at the Children's Hospital, Carlton, at 8.15 p.m..

## NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

CLOUSTON, KATHLEEN, M.B., Mast. Surg., 1921 (Univ. Sydney), Taralga.

ROBERTS, JOHN LESLIE, M.B., 1922 (Univ. Sydney), Lismore.

# University Intelligence.

## PROFESSIONAL EDUCATION IN MEDICINE.

THE following resolutions dealing with professional education have been passed by the General Medical Council on May 25 and 26, 1922, and will come into operation on January 1, 1923. These resolutions have been forwarded to the three Australian Universities with Faculties of Medicine in order that the curricula may be modified in conformity with them.

### Registration.

Every medical student at the commencement of his studies should be registered in the Medical Students' Register in the manner and under the conditions prescribed by the Council.

### Pre-Registration Examinations in Chemistry and Physics.

Before registration as a student or commencement of the regular medical curriculum every person shall be required to pass, in addition to an approved examination in general education, an examination or examinations conducted or approved by one of the licensing bodies in the following subjects:

(1) Physics (theoretical and practical), including the elementary mechanics of solids and fluids, the elements of heat, light, sound, electricity and magnetism. (This course should not include bio-physics or the clinical application of physics, which are to be taken in the medical curriculum.)

(2) Chemistry (theoretical and practical). The elements of the science. (This course should not include bio-chem-

istry, pharmacological chemistry or the clinical applications of chemistry, which are to be taken in the medical curriculum.)

### Medical Curriculum.

With regard to the course of study and examinations which persons desirous of qualifying for the medical profession shall go through in order that they may become possessed of the knowledge and skill requisite for the efficient practice of medicine, surgery and midwifery, the Council recommends as follows, *viz.*:

The period of professional study between the date of registration as a medical student and the date of the Final Examination for any Diploma which entitles its holder to be registered under the *Medical Acts*, should be a period of certified study during not less than five academic years, in the last three years of which clinical subjects shall be studied.

In every course of professional study and examinations the following subjects should be included:

(1) *Elements of General Biology.*—A course of instruction including practical work in the fundamental facts of vegetable and animal structure, life history and function and an introduction to the study of embryology. (The course of instruction may be taken before registration.)

(2) *Chemistry, Physics and Biology.*—Instruction in these subjects in their application to medicine.

(3) *Human Anatomy and Human Physiology.*—These courses should include (*a*) dissection of the entire body, (*b*) histology, (*c*) elements of human embryology, (*d*) biochemistry and bio-physics.

(4) *Elementary Bacteriology.*—A course in this subject should be taken before the student undertakes his regular clinical appointments [(8) (*ii.*); (9) (*ii.*)]

(5) *Pathology.*—Courses of instruction in (*a*) general and special pathology and morbid anatomy, (*b*) clinical pathology. Each student should be required to have received practical instruction in the conduct of autopsies and to have acted as a *post mortem* clerk in at least ten cases.

(6) *Pharmacology and Materia Medica, including Pharmacological Chemistry.*—A course including practical work should be taken concurrently with courses of clinical instruction.

(7) *Forensic Medicine, Hygiene and Public Health.*—Courses of instruction in these subjects should be taken concurrently with the later stages of clinical instruction.

(8) *Medicine, including Applied Anatomy and Physiology, Clinical Pathology and Therapeutics*, comprising:

(*i.*) A course of systematic instruction in the principles and practice of medicine.

(*ii.*) A medical clinical clerkship of six months, of which at least three months must have been spent in the hospital wards.

(*iii.*) Lectures or demonstrations in clinical medicine and attendance on general in-patient and out-patient medical practice during seven terms, which may be concurrent with the terms prescribed under (9) (*iv.*).

(*iv.*) Instruction in applied anatomy and physiology and in clinical pathology.

(*v.*) Instruction in therapeutics and prescribing, including pharmacological and physical therapeutics and the methods of treatment by vaccines and sera.

(*vi.*) Instruction in the following subjects, *viz.*, (*a*) children's diseases, (*b*) acute infectious diseases ("fevers"), (*c*) tuberculosis, (*d*) mental diseases, (*e*) diseases of the skin, (*f*) theory and practice of vaccination.

(9) *Surgery, including Applied Anatomy and Physiology and Clinical Pathology*, comprising:

(*i.*) A course of systematic instruction in the principles and practice of surgery.

(*ii.*) A surgical dressership for a period of six months, of which at least three months must have been spent in the hospital wards.

(*iii.*) Practical instruction in surgical methods, including mechano-therapeutics.

(*iv.*) Lectures or demonstrations in clinical surgery and attendance on general in-patient and out-patient surgical practice during seven terms, which may be concurrent with the terms prescribed under (8) (*iii.*).

(*v.*) Instruction in the administration of anæsthetics, the candidate being certified to have administered anæsthetics on at least ten occasions.

(*vi.*) A course of instruction in operative surgery.

(*vii.*) Instruction in applied anatomy and physiology and clinical pathology.

(*viii.*) Instruction in the following subjects, *viz.*, (*a*) diseases of the eye, refraction, use of ophthalmoscope; (*b*) diseases of the ear, throat and nose, use of otoscope, laryngoscope and rhinoscope; (*c*) radiology; (*d*) venereal diseases; (*e*) orthopædics, if this is not included in the course of surgery or of surgical methods.

(10) *Midwifery and Diseases of Women.*—Instruction during a period of at least two terms, comprising:

(*i.*) Courses of systematic instruction in the principles and practice of obstetrics and gynæcology.

(*ii.*) Lectures or demonstrations in clinical obstetrics and gynæcology and attendance on in-patient and out-patient gynæcological practice.

(*iii.*) Instruction in the following subjects, *viz.*, (*a*) antenatal conditions, (*b*) infant hygiene.

(*iv.*) Every student should, after attending the courses of systematic instruction in the principles and practice of surgery and of obstetrics, give continuous attendance on obstetrical hospital practice under the supervision of a competent officer for a period of three months, during one month of which at least he should perform the duties of an intern student in a lying-in hospital or ward. He should attend during the period twenty cases of labour under adequate supervision. Extern or district maternity work should not be taken until the student has personally delivered at least five cases in the lying-in hospital or ward to the satisfaction of his teacher.

A certificate of having attended twenty cases of labour should state that the student has personally attended each case during the course of labour, making the necessary abdominal and other examinations under the supervision of the certifying officer, who should describe his official position and state how many of the twenty cases were conducted in hospital.

### Additional Resolutions.

(*a*) That throughout the whole period of study the attention of the student should be directed by his teachers to the importance of the preventive aspects of medicine.

(*b*) That each licensing body should make adequate arrangements for the effective correlations of the several subjects of study throughout its curriculum.

(*c*) That the teaching of anatomy and physiology should include as a regular part of the courses the demonstration on the living human body of structure and function.

(*d*) That the curriculum should be so arranged that a minimum period of three years shall in every case be available for study after the completion by the student of the professional examinations in anatomy and physiology held at the close of the second year.

(*e*) That the curriculum should be so framed as to afford sufficient opportunities for the study during the last three years of the course of physics, chemistry, biology, anatomy and physiology in their practical applications to medicine, surgery and midwifery and that the student's knowledge of these applications should be subject to test in the Final Examination.

(*f*) That before the student is admitted to his clinical appointments he should have received practical instruction in clinical methods and in the recognition and interpretation of physical signs.

(*g*) That instruction should be given in the courses of forensic medicine and public health or otherwise on the duties which devolve upon practitioners in their relation to the State and on the generally recognized rules of medical ethics. Attention should be called to all notices on these subjects issued by the General Medical Council.

### Recommendations.

In addition, the General Medical Council has passed recommendations dealing with professional examinations in medicine as follows:

1. In order to secure due continuity and sequence in medical study, two or more professional examinations in

the earlier subjects should be held antecedently to the Final Examination in medicine, surgery and midwifery.

2. Three years at least should intervene between date of passing the professional examination in anatomy and physiology and that of admission to the Final Examination in medicine, surgery and midwifery.

3. A candidate remitted in any subject of a professional examination should, before he is re-admitted to examination therein, be required to produce satisfactory evidence that he has during the interval of remission pursued the study of the subject in which he was rejected. Candidates who obtain less than 30% of the marks in any subject should be remitted for a longer period than three months.

4. In all the professional examinations sufficient time should be assigned to practical work, in order to test the thoroughness of the candidate's knowledge and to encourage practical methods of study.

5. Candidates in all their examination work should be carefully supervised.

6. Two examiners should always participate in the oral examination of a candidate, except in subordinate parts of practical examinations.

7. In all written examinations the questions in each subject should be submitted for the approval of all the examiners in that subject.

8. In all written examinations an average of at least half an hour should be allowed for a candidate to answer each question.

9. It is desirable that examiners and in particular those for the Final Examination in medicine, surgery and midwifery, should be appointed or re-elected for at least three consecutive years.

10. Whatever may be the system of marking, the percentage for a pass in each subject should be not less than fifty.

11. In the regulations for the several examinations it should be provided that examiners in assessing marks be empowered to take into account the duly attested records of the work done by the candidate throughout his course of study in the subject of the examination.

12. The Final Examination in medicine, surgery and midwifery, with the exception of the clinical and practical examination in midwifery and gynæcology, must not be passed before the close of the fifth academic year of medical study.

13. The three portions of the Final Examination in medicine, surgery and midwifery should not be further subdivided into sections which may be entered for or passed separately.

14. Compensation in respect of marks as between the three different portions of the Final or Qualifying Examination, *viz.*, medicine, surgery and midwifery, is contrary to the intention of the *Medical Act, 1886*.

15. The Final Examination should include clinical and practical examinations in midwifery and gynæcology.

16. The clinical examination in medicine, surgery and midwifery should be held in properly equipped hospitals or examination halls, well provided with suitable patients.

17. In the examinations in clinical medicine at least one hour and in clinical surgery at least half an hour should be allowed to the candidate for the examination of and report on his principal case.

18. In medicine, in surgery and in midwifery no candidate should be allowed to pass who fails to obtain 50% of the aggregate marks assigned to the whole examination or who fails to obtain 50% of the marks assigned to the clinical examination or who fails to obtain 40% of the aggregate of the marks assigned to the written and oral examinations.

In midwifery, where a clinical examination is not held, the duly attested records of the work done by the candidate in clinical midwifery must be presented to the examiners for assessment in the Final Examination and no candidate should be allowed to pass who fails to obtain 50% of the aggregate marks assigned to clinical and practical midwifery and gynæcology.

19. The Final Examination should include examination of secretions, the testing of urine, clinical microscopy and prescription writing and there should always be an oral examination in medicine, surgery and midwifery, which should include an examination on pathological specimens.

20. At the Final Examination each candidate should be submitted to a practical and oral examination in pathology (macroscopic and microscopic), unless this has been included in a professional examination preceding the Final Examination.

21. Whatever be the method of entry for the Final Examination, all candidates should be required to complete the three portions of the Final Examination within a period of nineteen months.

# Naval and Military.

## APPOINTMENTS.

THE undermentioned promotions, changes, etc., have been notified in the *Commonwealth of Australia Gazette*, Nos. 62, 66, 68, 70, 71 and 77, of August 10, 24 and 31 and September 7, 14 and 21, 1922:

### Permanent Naval Forces of the Commonwealth.

SEA-GOING FORCES.

*Antedating the Seniority of an Officer.*

THE seniority of CHRISTOPHER BASIL HATTON BOAKE in the rank of Surgeon-Lieutenant (D) (on probation) is antedated to 17th January, 1922.

*Confirmation in Rank.*

SURGEON-LIEUTENANT (on probation) WILLIAM EDWARD JOHN PARADICE, M.B., Ch.M., is confirmed in the rank of Surgeon-Lieutenant, with seniority in rank of 1st August, 1921.

*Resignation.*

THE resignation of SURGEON-LIEUTENANT-COMMANDER CARLTON ATKINSON ELLIS, F.R.C.S., is accepted, dated 31st July, 1922.

*Transfer to Retired List.*

SURGEON-LIEUTENANT ERNEST SYDNEY GEORGE KILLEN VANCE, M.B., is transferred to the Retired List, dated 6th November, 1921.

### Citizen Naval Forces of the Commonwealth.

ROYAL AUSTRALIAN NAVAL RESERVE.

*Promotion.*

SURGEON-LIEUTENANT FREDERICK GLOVER NEASON STEPHENS, M.B., F.R.C.S., is promoted to the rank of Surgeon Lieutenant-Commander, dated 1st July, 1922.

*Appointments.*

CARLTON ATKINSON ELLIS, F.R.C.S., is appointed Surgeon-Lieutenant-Commander as from 1st August, 1922, with seniority in rank of 4th August, 1920, and is appointed District Naval Medical Officer, Victoria, as from the 1st September, 1922, with the acting rank of Surgeon-Commander whilst holding this appointment.

SURGEON-LIEUTENANT-COMMANDER PERCY GERALD SHELTON, M.B., B.S., is appointed Sub-District Naval Medical Officer, Port Melbourne, dated 1st September, 1922.

*Termination of Appointments.*

THE temporary appointments of SURGEON-COMMANDERS (RETIRED LIST) HARRY PAYNTER SLOGGETT and WILLIAM ARTHUR JAMES as District Naval Medical Officer, Victoria, and Sub-District Naval Medical Officer, Port Melbourne, respectively, are terminated, dated 31st August, 1922.

### Australian Military Forces.

FIRST MILITARY DISTRICT.

*Australian Army Medical Corps.*

LIEUTENANT-COLONEL F. C. WOOSTER, D.S.O., to be supernumerary to the establishment of Lieutenant-Colonels with pay and allowances of Major, 1st July, 1922.

*Reserve of Officers.*

CAPTAIN J. A. SHANASY, M.C., is transferred from the Reserve of Officers, Third Military District, 1st August, 1922.

SECOND MILITARY DISTRICT.

*Australian Army Medical Corps.*

THE provisional rank of LIEUTENANT-COLONEL R. DICK is confirmed.

*Australian Army Medical Corps Reserve.*

HONORARY CAPTAIN L. C. WEBSTER is transferred from the Australian Army Medical Corps Reserve, Sixth Military District, 1st August, 1922.

THE resignation of HONORARY CAPTAIN J. H. PEEK of his commission is accepted, 1st March, 1921.

THIRD MILITARY DISTRICT.

*Australian Army Medical Corps.*

MAJOR J. K. ADEY, O.B.E., is transferred to the Unattached List, 1st July, 1922.

MAJOR R. S. WHITFORD and CAPTAIN F. J. B. MILLER are transferred to the Reserve of Officers, 1st July, 1922.

MAJOR (provisionally) C. C. MARSHALL is transferred to the Reserve of Officers and to be Captain, 1st July, 1922.

CAPTAINS (provisionally) J. A. TROUP, F. W. TATE and H. I. ROBINSON are transferred to the Australian Army Medical Corps Reserve and to be Honorary Captains, 1st July, 1922.

MAJOR C. R. MERRILLEES is appointed from the Reserve of Officers and to be Captain, 1st July, 1922.

CAPTAIN G. E. COLE, D.S.O., is transferred to the Reserve of Officers and to be Major, 1st July, 1922.

CAPTAIN (provisionally) A. W. SHUGG is transferred to the Australian Army Medical Corps Reserve, Sixth Military District, and to be Honorary Captain, 1st July, 1922.

LIEUTENANT-COLONEL B. QUICK, D.S.O., relinquishes the command of tthe Second Field Ambulance, 30th June, 1922, and is transferred to the Unattached List, 1st July, 1922.

MAJOR R. W. CHAMBERS, D.S.O., is transferred to the Unattached List, 1st July, 1922.

CAPTAIN S. CRAWCOUR is transferred to the Reserve of Officers, 1st July, 1922.

CAPTAIN (provisionally) M. JACOBS is transferred to the Australian Army Medical Corps Reserve and to be Honorary Captain, 1st July, 1922.

THE resignations of LIEUTENANTS C. H. HEMBROW and L. V. DARBY of their provisional appointments are accepted, 30th June, 1922.

LIEUTENANT-COLONEL E. R. WHITE and LIEUTENANT-COLONELS (provisionally) J. J. MCMAHON, N. L. SPEIRS and W. W. W. CHAPLIN to be supernumerary to the establishment of Lieutenant-Colonels, with pay and allowances of Major, 1st July, 1922.

MAJORS (HONORARY LIEUTENANT-COLONELS) D. D. CADE, D.S.O., and E. W. GUTTERIDGE, and MAJOR E. CHAMPION to be supernumerary to the establishment of Majors, with pay and allowances of Captain, 1st July, 1922.

*Reserve of Officers.*

CAPTAIN A. J. BOTHAMLEY is transferred to the Reserve of Officers, Sixth Military District, 1st July, 1922.

CAPTAIN J. A. SHANASY, M.C., is transferred to the Reserve of Officers, 1st Military District, 1st August, 1922.

THE provisional appointment of CAPTAIN G. H. BRANDIS is terminated under the provisions of Section 16 of the *Defence Act*, 22nd August, 1922.

FOURTH MILITARY DISTRICT.

*Australian Army Medical Corps.*

LIEUTENANT-COLONEL H. H. E. RUSSELL, O.B.E., relinquishes the command of the Seventeenth Field Ambulance, 30th June, 1922, and is transferred to the Unattached List, 1st July, 1922.

LIEUTENANT-COLONEL A. R. CLAYTON, D.S.O., MAJORS C. CORBIN and F. GOLDSMITH and CAPTAINS K. N. STEELE and C. T. Turner, M.C., are transferred to the Unattached List, 1st July, 1922.

SIXTH MILITARY DISTRICT.

*Australian Army Medical Corps Reserve.*

CAPTAIN (provisionally) A. W. SHUGG is transferred from the Australian Army Medical Corps, Third Military District, and to be Honorary Captain, 1st July, 1922.

HONORARY CAPTAIN L. C. WEBSTER is transferred to the Australian Army Medical Corps Reserve, Second Military District, 1st August, 1922.

# Correspondence.

## THE BEHAVIOUR OF TETHELIN.

SIR: The review of the article on tethelin by J. C. Drummond and R. K. Cannan, which appeared in your issue of September 9, contains several errors of statement which are in part attributable to the authors of the article and in part to the abbreviation of the article for the purposes of review. A detailed reply to the criticisms of Drummond and Cannan will shortly appear elsewhere, but as that may possibly not be seen by some of your readers, I am taking this opportunity to point out a few of the more serious inaccuracies in Drummond and Cannan's article.

In the first place, your review states that: "The authors have made a chemical examination of several samples of material prepared as directed." Unfortunately, this is not the case. They did not prepare the material "as directed," but by an "improved" process of their own which I long ago discarded and never published, because I found that it involved far-reaching decomposition of the tethelin. Instead of employing an absolutely dry atmosphere of air during the preparation of tethelin, as I directed, they employed an atmosphere of carbon dioxide, which induces hydrolysis of the tethelin with easily demonstrable alteration of its physiological action. It was this decomposed material which Drummond and Cannan employed and, very naturally, they found that in some respects it resembled the material described by me, while in others it did not.

Your review goes on to state that: "The authors submitted the papers of Brailsford Robertson to Professor Karl Pearson and under the direction of the latter Mr. H. Soper prepared a report on the statistical methods. From this report it would appear that the curves failed to reproduce the characters of individual growth." As a matter of fact, the report is not included in their article and, beyond a vague atmosphere of indiscriminate criticism, no definite defect of method is alleged, except the fact that some of the animals in each experimental group differed by a maximum of three days in age from others. On this ground it is contended that the average weights do not represent the true average at any given age. Now, at twenty to thirty days of age a difference of three days between the ages of different animals might be a sufficient difference to introduce a certain degree of uncertainty into the measurements, but in my investigations animals are not employed for experimental purposes until they are thirty-five days old and the maximum effects of tethelin are displayed ten weeks later, namely, at fifteen weeks or one hundred and five days of age. I think that no person possessed of common sense will regard very seriously a criticism founded upon the lack of comparability of mice which are one hundred and five days old with other mice with differ from them in age by three days at the most and that only in a small proportion of instances. Such a contention would be indefensible, even if the animals were growing rapidly at one hundred and five days of age, but when it is recollected that at that age they are practically adult and therefore growing very slowly, the absurdity of this criticism is manifest.

Not content with this, however, Drummond and Cannan submit a variety of criticisms of their own devising, aimed at the statistical methods which I employ. These criticisms are based on the one hand upon misapprehension of statistical methods and on the other upon an indefensible manipulation of my published figures which creates a very unfavourable impression of their controversial methods. However, these will be adequately dealt with elsewhere.

The review states that: "Finally, the authors submitted a group of mice to the action of tethelin." Unfortunately, again, this was not the case. Instead of employing tethelin in their feeding experiments, they employed dried anterior lobe of the pituitary gland, notwithstanding the fact that their own experiments admittedly confirmed my statement of the susceptibility of tethelin to oxidation upon exposure to air in the presence of moisture. They conclude, upon the basis of an experiment with ten animals for a period of nine weeks, that administration of anterior lobe of the pituitary gland has no effect upon the growth of animals. In this they are not only at variance with my findings, but with those of Schafer, Cushing, Aldrich, Wulzen, Maxwell, Goetsch, Marinus, Pearl and especially the recent striking

observations of Uhlenhuth. In the course of my own experiments I have observed the effects of tethelin upon the growth of one hundred and forty animals and of fresh anterior lobe tissue upon seventy animals. The probable error of every estimate of weight is known and the probability of the observed deviations being accidental has been reduced to a vanishing magnitude. My observations upon the effects of anterior lobe tissue coincide with the results of previous and subsequent workers and have extended over the whole duration of the life of the animals. Nevertheless, I do not doubt the accuracy of Drummond and Cannan's observations, but I refer them to the destruction of tethelin incurred by drying the pituitary tissue with exposure to air. In preparing tethelin I avoided this by covering the tissue with anhydrous salts and subsequently excluding air, or only admitting it when thoroughly deprived of moisture and at a low temperature.

Yours, etc.,
T. BRAILSFORD ROBERTSON,
Professor of Physiology and Biochemistry.
University of Adelaide,
September 18, 1922.

## Proceedings of the Australian Medical Boards.

### QUEENSLAND.

THE undermentioned have been registered under the provisions of the *Medical Act of 1867* as duly qualified medical practitioners:

BRAKE, CLIFFORD EBROL, M.B., Ch.B., 1922 (Univ. Sydney), Toowoomba Hospital.
CARTER, ALEXANDER, M.B., Ch.M., 1921 (Univ. Sydney), Toowoomba Hospital.
FORD, JOHN WILLIAMS, M.B., 1918 (Univ. Sydney), Wynnum South.
GALLAGHER, WILLIAM PATRICK, M.B., 1922 (Univ. Sydney), Brisbane.
LOWSON, JAMES PRAIN, M.B., Ch.B., 1906, M.D., 1911 (Univ. Edinburgh), Brisbane.
MCCARTHY, EDWARD FRANCIS, M.B., Ch.M., 1921 (Univ. Sydney), Malanda.
SMITH, ERNEST BRUCE, M.B., Ch.M., 1920 (Univ. Sydney), Isisford.
STUART, ADAH ANNIE MACDONALD, M.B., Ch.M., 1922 (Univ. Sydney), Rockhampton.

## Medical Appointments.

DR. C. J. TONKIN (B.M.A.) has been appointed Certifying Medical Practitioner at Sunshine, Victoria, under the provisions of the *Workers' Compensation Act, 1915*.

* * * *

DR. C. D. MACCARTHY has been appointed Public Vaccinator at Omeo, Victoria.

* * * *

THE COMMISSION OF PUBLIC HEALTH IN VICTORIA has appointed the surgeries of the undermentioned as places for public vaccination: DR. R. D. FISHER (B.M.A.) at Sorrento, DR. C. F. MACDONALD (B.M.A.) at Warracknabeal, DR. B. VAN SOMEREN (B.M.A.) at Bright, DR. S. M. WILCOX at Macarthur.

* * * *

DR. W. A. MACKEY and DR. K. F. POTTS have been appointed Junior Resident Medical Officers at the Perth Hospital, Western Australia.

* * * *

DR. K. F. VICKERY (B.M.A.) has been appointed Medical Officer of Health, Hunter River Combined District, New South Wales.

## Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii..

SYDNEY HOSPITAL: Honorary Pathologist.

## Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

| BRANCH. | APPOINTMENTS. |
|---|---|
| NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney | Australian Natives' Association<br>Ashfield and District Friendly Societies' Dispensary<br>Balmain United Friendly Societies' Dispensary<br>Friendly Societies Lodges at Casino<br>Leichhardt and Petersham Dispensary<br>Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney<br>Marrickville United Friendly Societies' Dispensary<br>North Sydney United Friendly Societies<br>People's Prudential Benefit Society<br>Phœnix Mutual Provident Society |
| VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne | All Institutes or Medical Dispensaries<br>Australian Prudential Association Proprietary, Limited<br>Manchester Unity Independent Order of Oddfellows<br>Mutual National Provident Club<br>National Provident Association |
| QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane | Brisbane United Friendly Society Institute<br>Stannary Hills Hospital |
| SOUTH AUSTRALIA: Honorary Secretary, 12, North Terrace, Adelaide | Contract Practice Appointments at Renmark<br>Contract Practice Appointments in South Australia |
| WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth | All Contract Practice Appointments in Western Australia |
| NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington | Friendly Society Lodges, Wellington, New Zealand |

## Diary for the Month.

SEPT. 30.—Victorian Branch, B.M.A.: Election of Two Members to Federal Committee.
OCT. 1.—Victorian Branch, B.M.A.: Election of Representatives of Divisions.
OCT. 3.—New South Wales Branch, B.M.A.: Council (Quarterly).
OCT. 4.—Victorian Branch, B.M.A.: Branch.
OCT. 6.—Queensland Branch, B.M.A.: Branch.
OCT. 6.—Annual Meeting of the Delegates of the Local Associations with the Council of the New South Wales Branch, B.M.A.: First Day.
OCT. 7.—Annual Meeting of the Delegates of the Local Associations with the Council of the New South Wales Branch, B.M.A.: Second Day.
OCT. 10.—New South Wales Branch, B.M.A.: Ethics Committee.
OCT. 11.—Western Australian Branch, B.M.A.: Council.
OCT. 11.—Melbourne Pædiatric Society.
OCT. 12.—Victorian Branch, B.M.A.: Council.
OCT. 13.—New South Wales Branch, B.M.A.: Clinical Meeting.
OCT. 13.—Queensland Branch, B.M.A.: Council.
OCT. 13.—South Australian Branch, B.M.A.: Council.

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